VOL. LXX JANUARY, 1927 NEW SERIES VOL. 23, NO. 4

# JOURNAL of the American Veterinary Medical Association

FORMERLY

AMERICAN VETERINARY REVIEW

(Original Official Organ U. S. Vet. Med. Ass'n.)

EDITED AND PUBLISHED FOR

The American Veterinary Medical Association

## CONTENTS



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# JOURNAL

## OF THE

# American Veterinary Medical Association

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H. Preston Hoskins, Secretary-Editor, 716 Book Building, Detroit, Mich.





Vol. LXX, N. S. Vol. 23 January, 1927 No. 4


## HAPPY NEW YEAR

The Secretary-Editor takes this opportunity to acknowledge the many season's greetings sent him the past month. From the middle of November to the middle of December, upwards of two thousand pieces of mail were received at the A. V. M. A. office. A large number of these were remittances for dues and many of the later arrivals were accompanied by greetings of some sort—in many cases just a few words written on the slip accompanying the check. Each and every one has been appreciated and enjoyed. It has been impossible to acknowledge each one individually, so it must be done collectively. The year 1926 saw conditions much improved for the profession generally and there is much to be thankful for. The year upon which we are just entering promises much and even more if we will measure up to our opportunities. Let each one of us work just a little harder to make 1927 a really

HAPPY NEW YEAR!

## WHAT DID YOU DO WITH YOURS?

When the statements for the 1927 dues were being prepared for mailing, it was found that the letters would take an additional enclosure without extra postage. In some we inserted a reprint, in others an announcement concerning liability insurance, in others a blank application for membership, etc. No letter was included to direct attention to the enclosure or to tell what to do with it. However, a number of members promptly put their enclosures to work, as events later showed.

One veterinarian received a reprint of the article by Dr. Utter, "Pure Milk Absolute Necessity for Children." He immediately wrote out a check for his dues and enclosed it with a letter requesting a sufficient number of copies for the members of his local board of health. Incidentally he stated that his city was in urgent need of a more stringent dairy inspection ordinance and he wanted the health authorities to read Dr. Utter's article.

Some of the applications for membership were put to work. We are listing ten applications this month, against four for the same month last year. We are giving second listing to eight applicants, against four for the same month last year. A healthy increase, but not as many as we ought to have.

Now then, upwards of three thousand enclosures were mailed. Some have been heard from. Others have not. What did you do with yours?

## PHILADELPHIA MEETING DATES SELECTED

At the meeting of the Executive Board, held in Chicago, December 1, 1926, it was decided to hold the Philadelphia meeting on the four days beginning Tuesday, September 13, 1927. A tentative outline of the program was agreed upon as follows:

| | Monday Sept. 12 | Tuesday Sept. 13 | Wednesday Sept. 14 | Thurdsay Sept. 15 | Friday Sept. 16 |
|---|---|---|---|---|---|
| Morning | Meetings of Committees | Opening Session | Sectional Meetings | Sectional Meetings | Clinic |
| Afternoon | Meeting of Executive Board | General Session | General Session | General Session | Clinic |
| Evening | State Association Conference | Alumni Meetings. President's Reception | Banquet | General Session — Papers | |

## A REMINDER

Dues for 1927 are due. If you have not already forwarded a remittance for these dues, kindly do so as soon as possible and thereby relieve the Association of the expense of sending you another notice. By the time this number of the JOURNAL is received by our members, approximately forty per cent will have received their 1927 membership cards. Do your part by helping us to finish this job by February 1.

## APPOINTMENTS

In another part of this issue will be found the organization of the A. V. M. A. for 1926-1927. It was necessary for President Sigler to make no less than 129 appointments of various kinds this year—no small task. All appointees have been notified and the majority have indicated their acceptance of the assignment. Quite a number of new names appear in the list. An infusion of new blood in any organization invariably is followed by renewed activity. Let the slogan of all committees and resident secretaries be: "Lets' get to work!"

## CONSPICUOUS MENTION

It is rather unusual for the President of the United States to refer to a veterinary activity in his annual message to Congress. However, this year, President Coolidge touched upon the subject of the eradication of bovine tuberculosis in the following words:

> . . . Another preventive measure of great economic and sanitary importance is the eradication of tuberculosis in cattle. Active work is now in progress in one-fourth of the counties of the United States to secure this result. Over 12,000,000 cattle have been under treatment, and the average degree of infection has fallen from 4.9 per cent to 2.8 per cent. The federal government is making substantial expenditures for this purpose. . . .

## APPLICATIONS FOR MEMBERSHIP

(See October, 1926, JOURNAL)

FIRST LISTING

BURINGTON, IRVIN O. Marie Apts., South St. Paul, Minn.
D. V. M., McKillip Veterinary College, 1920
Vouchers: G. E. Totten and Irvin Owens.

CONLIN, WILLIAM JEROME 608 Lincoln Ave., Apt. 105-C, St. Paul, Minn.
D. V. M., Cornell University, 1920
Vouchers: G. E. Totten and Irvin Owens.

GOODING, CLOYDE LEE 509 Grand Ave., So. San Francisco, Calif.
D. V. M., Colorado Agricultural College, 1926
Vouchers: W. J. Stewart and Frank Hurlbut.

HATFIELD, GEORGE RAIFORD Monroe, Ga.
D. V. M., Georgia State College of Agriculture, 1926
Vouchers: R. M. Walsh and A. G. G. Richardson

KINNEY, WILLIAM GRAHAM 2420 4th Ave., Seattle, Wash.
D. V. M , Michigan State College, 1925
Vouchers: E. A. Ehmer and Ward Giltner.

SIMMONS, GEORGE M. 2500 16th St., San Francisco, Calif.
D. V. M., Iowa State College, 1925
Vouchers: Oscar J. Kron and M. A. Emmerson.

STOUT, FRANK Mendon, Ill.
Terre Haute Veterinary College, 1913
Vouchers: Frank H. Brown and W. H. Welch.

SWENSON, LLOYD ALEXANDER Sherburn, Minn.
D. V. M., Chicago Veterinary College, 1917
Vouchers: H. C. H. Kernkamp and W. L. Boyd.

WHITMORE, WILLIAM WESLEY 6204 So. Park Ave., Chicago, Ill.
D. V. M., Chicago Veterinary College, 1915
Vouchers: H. M. Springer and J. M. Handley.

YOUNG, GUY F. Marshall, Minn.
D. V. M., Kansas City Veterinary College, 1918
Vouchers: W. L. Boyd and E. A. Hewitt.

## Applications Pending

SECOND LISTING

Davidson, Arthur C., 178 Hailesboro St., Gouverneur, N. Y.
Hell, Henry, Wilton Junction, Iowa.
Reed, Francis Irving, Morris, N. Y.
Roberts, Henry Powell, Edgeley, No. Dak.
Sharp, Milton R., State College, N. Mex.
Spevack, Victor, 1665 Bedford Ave., Brooklyn, N. Y.
Starr, Leland E., 56 Hull Ave., Freehold, N. J.
Wisinicky, Walter, 1031 Williamson St., Madison, Wis.

## Reinstated

Hinkle, Truman B., Sulphur Springs Stock Farm, Ashley, Ohio.

The amount that should accompany an application filed this month is $10.00, which covers membership fee and dues to January 1, 1928, including subscription to the JOURNAL.

## COMING VETERINARY MEETINGS

Nevada State Veterinary Association. Reno, Nev. Jan. 3, 1927. Dr. Edward Records, Secretary, University of Nevada, Reno, Nev.

Pennsylvania Conference of Veterinarians, University of. Philadelphia, Pa. Jan. 4-5, 1927. Dr. Louis A. Klein, Dean, 39th St. & Woodland Ave., Philadelphia, Pa.

California Veterinary Conference, University of. Davis, Calif. Jan. 4-5-6-7, 1927.

Washington Conference for Veterinarians, State College of. Pullman, Wash. Week of Jan. 10, 1927. Dr. E. E. Wegner, Dean, College Sta., Pullman, Wash.

Southeastern Michigan Veterinary Medical Association. Detroit, Mich. Jan. 12, 1927. Dr. H. Preston Hoskins, Secretary, 716 Book Bldg., Detroit, Mich.

Maine Veterinary Medical Association. Waterville, Me. Jan. 12, 1927. Dr. A. J. Neal, Secretary, 324 Essex St., Bangor, Me.

Kansas Veterinary Medical Association. Topeka, Kans. Jan. 12-13, 1927. Dr. Chas. W. Bower, Secretary, 1128 Kansas Ave., Topeka, Kans.

Minnesota State Veterinary Medical Association. Radisson Hotel, Minneapolis, Minn. Jan. 13-14, 1927. Dr. C. P. Fitch, Secretary, University Farm, St. Paul, Minn.

Cornell University, Nineteenth Annual Conference for Veterinarians at. Ithaca, N. Y. Jan. 13-14, 1927. Dr. V. A. Moore, Dean, N. Y. State Veterinary College, Ithaca, N. Y.

Ohio State Veterinary Medical Association. Columbus, Ohio. Jan. 13-14, 1927. Dr. W. R. Hobbs, Secretary, Ohio State University, Columbus, Ohio.

California State Veterinary Medical Association. Sacramento, Calif. Jan. 13-14-15, 1927. Dr. E. H. Barger, Secretary, Davis, Calif.

Mississippi State Veterinary Medical Association. Hattiesburg, Miss. Jan. 17-18, 1927. Dr. Wilbur McPherson, Secretary, Brookhaven, Miss.

Oklahoma State Veterinary Medical Association. Huckins Hotel, Oklahoma City, Okla. Jan. 17-18, 1927. Dr. C. H. McElroy, Secretary, Stillwater, Okla.

Indiana Veterinary Medical Association. Indianapolis, Ind. Jan. 18-19-20, 1927. Dr. R. H. Boyd, Secretary, 446 E. 10th St., Indianapolis, Ind.

Iowa Veterinary Association. Hotel Savery, Des Moines, Iowa. Jan. 18-21, 1927. Dr. E. R. Steel, Secretary, Grundy Center, Iowa.

Arkansas Veterinary Association. Hotel Marion, Little Rock, Ark. Jan. 19, 1927. Dr. Hubert Shull, Secretary, 414 W. 3rd St., Texarkana, Ark.

Colorado Veterinary Medical Association. Denver, Colo. Jan. 19, 1927. Dr. R. F. Bourne, Secretary, Fort Collins, Colo.

Pennsylvania State Veterinary Medical Association. Harrisburg, Pa. Jan. 19, 1927. Dr. H. R. Church, Secretary, Harrisburg, Pa.

Texas, State Veterinary Medical Association of. San Antonio, Texas. Jan. 19-20, 1927. Dr. D. Pearce, Secretary, Leonard, Texas.

Maryland State Veterinary Medical Association. Medical Hall, Baltimore, Md. Jan. 20, 1927. Dr. E. M. Pickens, Secretary, College Park, Md.

Michigan State College Short Course for Veterinarians. East Lansing, Mich. Jan. 24-28, 1927. Dr. Ward Giltner, Dean, East Lansing, Mich.

Wisconsin Veterinary Medical Association. Madison, Wis. Jan. 25-26-27, 1927. Dr. O. H. Eliason, Secretary, 226 W. Gilman St., Madison, Wis.

Missouri Special Course for Graduate Veterinarians, University of. Columbia, Mo. January 25-26-27-28, 1927.

Alabama Veterinary Medical Association and Short Course for Practitioners. Auburn, Ala. Jan. 31-Feb. 1-2-3-4-5, 1927. Dr. C. A. Cary, Secretary, Auburn, Ala.

Connecticut Veterinary Medical Association. Hartford, Conn. Feb. 2, 1927. Dr. Geo. E. Corwin, Secretary, 11 Warrenton Ave., Hartford, Conn.

Florida State Veterinary Medical Association. Gainesville, Fla. Feb. 7-8, 1927. Dr. A. L. Shealy, Secretary, University of Florida, Gainesville, Fla.

Ontario Veterinary Association. Prince George Hotel, Toronto, Ont. Feb. 9, 1927. Dr. H. M. LeGard, Secretary, 223 Main St. No., Weston, Ont.

Kansas State Agricultural College Conference for Veterinarians. Manhattan, Kans. Feb. 9-10, 1927. Dr. R. R. Dykstra, Dean, K. S. A. C., Manhattan, Kans.

Northwestern Ohio Veterinary Medical Association. Toledo, Ohio. Feb. 22, 1927. Dr. F. A. Lambert, Secretary, c/o Columbus Serum Co., Sta. C, Box 53, Columbus, Ohio.

## STATE BOARD EXAMINATIONS

Florida State Board of Veterinary Examiners. Gainesville, Fla. Jan. 11-12, 1927. Dr. A. L. Shealy, Secretary, University of Florida, Gainesville, Fla.

Texas State Board of Veterinary Medical Examiners. San Antonio, Texas. Jan. 17, 1927. Dr. R. G. Flowers, Secretary, 3rd & Main Sts., Fort Worth, Texas.

Arkansas Veterinary Examining Board. Old State House, Little Rock, Ark. Jan. 20, 1927. Dr. Joe H. Bux, Secretary, Old State House, Little Rock, Ark.

# RESEARCHES IN THE PATHOGENESIS OF MILK FEVER*

*By* M. L. AUGER

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*Translated by* C. J. MARSHALL

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*Milk fever is a manifestation of a hypoglycemic syndrome:* The symptomatology of milk fever is well known. This disease generally follows promptly after parturition. It is shown, most often, by a progressive torpor, bordering on a profound collapse, with a subnormal temperature. Before 1897, the prognosis was always grave, for the mortality, according to statistics of various authorities, was about fifty per cent. Beginning in 1897, the year when Schmidt[1] brought forth the treatment which bears his name, the losses fell to fifteen per cent. With the treatment of Anderson, rendered practical by the Evers invention (1901), the mortality abated much more and its rate since then has not been more than one to two per cent; when this treatment is used the patients are generally cured in a few hours, in a way that is surprisingly rapid. Milk fever is really peculiar; it is, in fact, a disease without prodromes or convalescence.

If the symptoms of milk fever are well known today, we know but little concerning its etiology and we are ignorant also, so to speak, of its pathogenesis and of the mysterious action of the peculiar treatment. For more than a hundred years milk fever has been classified as a morbid entity; numerous theories have been put forth regarding its nature, most of them reflecting the ruling medical doctrines of the times. Certain of these theories are evidently delusive but no experimental basis has been established; nothing has been found to account for the onslaught of the disease following accouchement, nor, on the other hand, its sudden cure by inflation of the udder.

The theory which we present in this article is not merely a deduction following a clinical examination of the disease; it is supported by precise experiments. Let us say at once that we consider milk fever to be a manifestation of a hypoglycemic syndrome. The first mention of a theory comparable to that

*Revue Générale de Médecine Vétérinaire, xxxv(1926), 415, pp. 353-384.

which we now endeavor to establish was given in November, 1923, by M. Neefs,[2] Military Veterinarian, in a communication to the Belgian Society of Veterinary Science. This colleague, in his account of the use of insulin, reported that a Canadian veterinarian (whose name, unfortunately, I do not know), struck by the similarity of symptoms observed in hypoglycemia experimentally produced by an injection of insulin and those which one recognizes in milk fever, had the idea that this disease could be due to hypoglycemia and he proved the fact by successfully treating a cow having milk fever with an injection of glucose, but this form of treatment, to our knowledge at least, has never been repeated. The veterinary publications, French and foreign, are mute on the subject, up to 1925. At the present time, two Swedish authors, Erick M. P. Widmark, professor of biologic chemistry, of the faculty of medicine, Lund, and Olof Carlens, are publishing the first results of their experiments on the manifestations of hypoglycemic syndromes and their relation to milk fever.[3] At the beginning of 1926 an English veterinarian, L. C. Maguire,[4] formulated a theory, without doubt totally different from that which we have sought to establish, but he credited nevertheless a great importance to hypoglycemia. In short, having carried on an experiment for several months, we[5] propose a new theory for milk fever which constitutes the foundation of this article. The experiments and conclusions of these authors and ours we will review in this preliminary study. We will recall, in the beginning, a few of the causes of milk fever universally admitted, thus giving us an opportunity of mentioning what we have perceived on the study of the hypoglycemic syndrome, explaining certain clinical conditions which have been baffling to not a few. We will then examine the theories put forth on the pathology of milk fever and review the study from a knowledge of the normal amount of glycogen of bovine animals. We shall consider the manifestations of hypoglycemia in bovines, the effect of Evers' treatment on gylcemia and the investigations on the percentage of sugar in the blood during the course of milk fever. Finally, we will show a coordination of all these data and the formulation of a theory on the pathology of milk fever.

## Etiology of Milk Fever

*The appearance of milk fever is based upon an abundance of milk secretion:* Delmer,[6] in his thesis on vitulary eclampsia, justly

draws attention to the fact that all the causes universally admitted demonstrate that an abundant lactation is the chief cause of this malady. This fact goes to support the theory that we put forth.

Cows which are heavy milkers being most often affected, it is easy to establish classifications of the bovine breeds. This can be done from the yield of milk as follows: The Hollandaise and sub-divisions; the Flemish, in particular, and the Normans, black-and-whites, etc. On the contrary the disease is exceptional in the races of southern France, which are inferior milkers, and the beef breeds. Among the breeds above mentioned those which have the aptitude for milk production the most marked are most subject to the disease. A distinguished practitioner of southern France, Mr. Bru,[7] has called attention to this fact and has shown that certain cows, especially heavy milkers, may contract the disease three or four times during their life. Milk fever affects, above all, cows during the age of maximum milk production. It is exceptional in cows with the first calf and most likely to appear after the third, fourth and fifth parturition, or between the ages of six and eleven years, when milk production is at its maximum.

When milking is suspended a certain time before parturition, milk fever appears more frequently than if milking is continued almost to the birth of the calf. This peculiarity has been mentioned by numerous practitioners. Experiments conducted in America and in France have shown that a rest of about sixty days (dry period) is necessary to obtain the maximum yield of milk. When this rest is for not more than forty days the production of milk during the period of lactation is reduced about thirteen per cent.[8] This rest, therefore, acts as a cause by favoring milk secretion.

Complete and repeated milkings, practiced after parturition, are considered by Hoyois[9] and by Bru[10] as capable of favoring the appearance of milk fever. The effects of mammary gymnastics on milk secretion are too well known to need further discussion. We will recall the old observation of Gunther and Felizet.[11] These investigators have mentioned that milk fever is observed more frequently in a dairy center than in a breeding section. In the first instance the calf is removed from its mother at birth in order that the maximum quantity of milk may be obtained; in the second case the calf is left with the mother. They concluded that the distress of the mother, incident to the separa-

tion from the calf, could cause the disease. All later authors on the subject of milk fever have denied or ridiculed such a cause. The observations of Gunther and Felizet are correct but the interpretation which they gave is fanciful. Although massage of the udder by the calf favors functioning of the gland, it is probable that, in good milkers at least, the calf does not empty the udder as completely as can de done by a man milking. It has not been demonstrated by experimentation but in order to show the justification of our opinion let us note in passing that in good milkers which suckle their progeny the appetite of the calf is not sufficient to empty the udder and that in numerous observations, after having allowed the calf to satisfy its appetite, one may still obtain an appreciable amount of milk by hand milking. It is because the udder is emptied more completely by hand milking that the disease is favored.

It is also after an easy parturition that one sees the disease; the lacteal secretion is at its maximum; nothing of an infectious nature or otherwise is present to interfere with the activity of the udder. To this one can add another statement of Hoyois.[9] This author called attention to the fact that one never sees milk fever in a cow that contracts mastitis immediately after calving. This is accounted for by the fact that milk secretion is decreased if not wholly discontinued.

Intensive feeding in the weeks preceding parturition has been considered by many as a cause favorable to the appearance of milk fever. It increases milk secretion. Those who have blamed abundant feeding as a cause have neglected, unhappily, to make known to us the nature of the food suspected. Frequently milch cows are given considerable quantities of food rich in water (brewers' grains) but which in reality is of but little nutritive value. It is difficult to hold this theory against what Maguire[4] has recently brought forth. In support of his opinion he cites the practice of English breeders. They, in the six weeks before calving, give their animals nutritive concentrates and withhold all voluminous food having a restricted nutritive value; this method, which they call "steaming," increases the lacteal secretion and diminishes the cases of milk fever by an accumulation of glycogen and fat.

Other causes of milk fever have been suggested (pasture or stable, cold, atmospheric pressure, etc.) but do not appear, according to contradictory statements, to play an important part in the etiology of the disease. We will not discuss them.

Of all these causes the best established appears to be that milk fever is under the strict dependence of milk secretion. The better milkers cows are, the greater their aptitude to develop the disease.

### Insufficiency of the Pathogenetic Theories of Milk Fever

The nature of milk fever has always been mentioned as a disease of obscure pathogenesis and with a considerable number of theories. It is customary to arrange them under four headings as follows: Circulatory; infection; auto-intoxication; and anaphylaxis. I shall add a more recent theory, put forth abroad, which is based upon an insufficiency of certain endocrine glands.

We will not discuss the theory of infection; it was abandoned long ago. The circulatory theory (congestion, anemia, aremia of certain nerves) has but few followers; among them we will limit ourselves to citing the most recent: Seitter, 1910.[12] He thinks that in the course of milk fever there exists an alteration of certain vasomotor centers with a low blood pressure as a consequence. Insufflation of the udder acts in relieving the low blood pressure. Seitter demonstrates it experimentally by means of the apparatus of Riva-Rocci by recording the arterial pressure of chloralized cows and then inflating the udders. It has not been demonstrated that lowering of the arterial pressure is sufficient to cause the phenomenon of coma in a cow; moreover, the experiments of Widmark and Carlens and ourselves prove that in addition to an elevation of blood pressure (which we have not denied for a moment) the insufflation of air determines a notable augmentation of glycemia.

The theory of auto-intoxication has been in great repute for many years; it appears to coincide with the ruling medical doctrines; it still has followers. Auto-intoxication of hepatic or renal origin might be considered first. In the first case because the liver is the transforming and eliminating organ par excellence of the greater part of the poisonous substances which are elaborated in the course of nutrition or of the functioning of the organs. In the second case because the kidneys are the emunctory for a large number of toxic products. But the signs of hepatic insufficiency or renal lesions are far from being constant in the course of milk fever, and then, let us add in order to return to the major question, how explain an action so rapid as the Evers treatment? The theory of auto-intoxication of mammary origin was first considered. All practitioners having observed, as men-

tioned above, that milk fever is intimately connected with the functioning of the udder, nothing more natural than to think that such intense gymnastics should cause the disease; but all who from time to time have supported it, viz., Allemani, Gratia, Schmidt, Delmer, Hoyois, etc., are by no means in accord on its innermost mechanism. According to Gratia it is the new gland cells which undergo transformation into colostrum, delivering into the circulation large amounts of waste products, and so the emunctories (liver and kidneys) are not able to control the disease. Schmidt attributes a favorable action to the large mononuclears charged with fat globules, which appear normally in colostrum and which are known under the name of corpuscles of Donné. Delmer considers that intoxication is a result of resorption of the products of degenerated albuminous material; a proteolytic ferment is elaborated by the epithelial cells of the udder; it becomes active under the influence of the reflex excitation of the uterus and acts on the casein. Auto-intoxication of mammary origin appears difficult at first sight to admit; the role of this gland as an emunctory is negligible and its intervention in general metabolism in no case can be compared to that of the liver and kidneys. Still more recent investigations appear to contradict this hypothesis. Porcher and Panisset,[13] by an ingenious experiment, have shown that colostrum is only a milk modified by retention. Porcher[14] has studied carefully the phenomena of milk retention. He has shown that it is the lactose especially that is absorbed and that lactose is partially eliminated by the urine. Milk retention is accompanied then by lactosuria. This has great importance, let us note in passing, and permits us to explain lactosuria in cows affected with milk fever. Casein, to the contrary, in case of milk retention, is the object of qualitative modification, above all is not absorbed—or in infinitesimal quantity—but is only transformed. As to the fat content, resorption is effected by the mechanism of phagocytosis; one sees appearing in the retained milk large mononuclears charged with globules of fat to which Schmidt attributes great importance; they were observed by Czerny, by Weil and Thevenet, and are known as the corpuscles of Donné. Retention of milk is not always accompanied by other trouble, nor in all cases are the symptoms identical with those of milk fever. Besides, one can always inject milk or colostrum into the udder without altering the health but it is not so when milk or colostrum is introduced subcutaneously, as has been shown by Delmer; one then produces

the phenomena of shock, more or less marked, which facts are well known since the utilization of proteinotherapy and lacto-proteinotherapy.

One can still bring forth against the theory of auto-intoxication of mammary origin, if the above arguments have not been sufficient, clinical facts which are not without importance: Repeated milking after parturition should prevent development of milk fever, however, according to Hoyois and Bru, it favors its appearance; the occurrence of mastitis immediately after parturition, with the diminution of milk secretion, never coincides with the appearance of milk fever (Hoyois).

However fragile may be the hypothesis of the formation of an intramammary poison as the cause of milk fever, it is no less strange than the efficacious results that have followed the discovery of the Andersen-Evers treatment.

The theory of anaphylactic shock bears the mark of the critical mind of our Belgian colleague, Van Goidsenhoven[15]; it is too well known for discussion; as in the preceding theory one can reproach him with being too theoretical and make divers objections. Porcher has shown that in milk retention, just mentioned, the albumins were above all transformed but not absorbed, or only in an infinitesimal quantity; moreover, in causing anaphylactic shock experimentally by casein in animals of the bovine species, one should remember the symptoms of milk fever; but those of anaphylactic shock, if one believes the observations of Alexandrescu and Ciuca,[16] are all different from those of milk fever. The progressive torpor which is characteristic of the disease is not constant. In a word, none of the tests symptomatic of shock were produced; leucopenia, inversion of the leucocytic formula, faulty coagulation, bright-colored venous blood, lowering of the refraction index not having been demonstrated to be present in milk fever; we are still ignorant as to whether the subcutaneous injections of Besredka are an efficacious prophylaxis. In spite of these objections the theory of Van Goidsenhoven remains no less a seductive doctrine, which during the course of the last few years has coincided with the opinions of most veterinarians.

The existence, in the course of milk fever, of disturbance of the endocrine glands has been supported by various authors; especially Dryerre and Greig (1925)[17] and Maguire (1926).[4] We will set forth now only the theory of the first and later will discuss the theory of Maguire regarding the intervention of hypo-

glycemia. According to Dryerre and Greig, milk fever is due to parathyroid deficiency. These authors have observed that in the case of milk fever there was a lowered amount of calcium in the blood and one knows since the experiments of MacCallum, Collip,[18] etc., that the endocrine glands and in particular the parathyroids play an important role in the metabolism of calcium. But the lowering of the percentage of calcium in the blood is constant in the course of gestation; when this percentage descends too low it generally causes modifications of the skeleton, rarely spasmodic disturbances, never comatose conditions. The results of calcium deficiency in bovines are well known. In fact, in heavy milkers, in spite of heavy feeding, American breeders have observed the appearance of disease of the skeleton, of demineralization of bone due to the diminution of the percentage of calcium. The elaboration of milk has demanded a large quantity of mineral matter. Moreover, does this explain the Evers treatment?

On account of the failure of the theories proposed up to the present time to explain the pathology of milk fever one must continue to investigate. So, by correcting the comatose phenomenon observed in hypoglycemia and in milk fever we have thought that it might be due to an exhaustion of glucose from the blood. Before establishing this theory we will first obtain a general idea of the percentage of glycemia in bovines; this study will lead us to see that the percentage is always lower in milch cows and will explain to us the ease with which the syndrome of hypoglycemia can appear.

## Glycemia of Bovines

*The percentage of glycemia in milch cows is lower than the percentage of glycemia in bovines not producing milk:* Blood contains glucose; the percentage of glycemia is very variable, according to the species, animals, and the same animal at different times. These variations depend upon the amount of sugar in the food, the amount of glucose consumed. When food is rich in carbohydrates or in substance capable of being transformed into glucose and when the waste material of the body is low, the percentage of glycemia is increased; it is lowered, on the contrary, when the sugar consumption of the body is raised and when the food is low in nutritive value. In reality these oscillations in glycemia are little marked; when the percentage of glucose is increased it reaches its limit and is eliminated by the

kidneys. The limit is variable with the species and is not fixed even in the same individual. According to Ambard and Chabanier,[19] it varies with the condition of the kidneys (is raised in renal sclerosis) and with the glycemia; the more glycemia is increased the more the limit is raised, although restrained to a certain degree. In man it oscillates around 1.70 but may reach two grams per liter. When the stage of glycemia is lowered to a marked degree, grave symptoms, sometimes mortal, manifest themselves, constituting the syndrome of hypoglycemia. The minimum limit is not absolutely constant for all animals, but, if one recalls the numerous experiments with insulin, it is more fixed than the maximum. Such symptoms generally occur when the amount of sugar in the blood reaches 0.30 to 0.40 grams to the liter. Between the maximum and the minimum limits there exists an intermediary zone before symptoms will appear, in which the amount of glucose can oscillate without the manifestation of trouble.

In milch cows the consumption of glucose by the organism is much greater than in other animals. In fact, beyond the sugar necessary for nutrition and the functioning of the organs, there must be a considerable quantity for the elaboration of lactose by the cells of the mammary gland. The experiments of Porcher,[20] now classical, have shown that lactose in milk originates from the glucose in blood. One can easily understand that, in these animals, the consumption of glucose must be very great, increasing as milk production increases. An example will enable one to understand it. Lactose, one knows, is one of the most fixed elements in milk, averaging 50 gms. per liter. Widmark and Carlens have calculated that a cow giving 28 Kg. of milk per day must supply 60 gms. of glucose per hour and that all the sugar content of the blood must be renewed every quarter of an hour. The intensity of lactation must then have a great influence on the sugar content in blood, which is obtained from the food as observed above.

Facts have confirmed these deductions. Following Widmark and Carlens, we have practised blood tests in bovine animals. For such tests we have utilized the method of G. Bertrand and have generally used 20 gms. of blood; we have always taken two samples at the same time and the results given are the average of the two tests. Widmark and Carlens used the micrometric method of Bang; it has the great advantage of permitting the test with very small quantities of blood, 0.5 to 1.0 cc. This

method, valuable in man and small, laboratory animals, we have not used in the cow and goat where one can repeat taking 20 to 30 gms. of blood several times without inconvenience.

Let us remark at the beginning that normal glycemia is a little lower in the cow and goat than in other animals. Porcher has mentioned this fact.[20] Widmark and Carlens have found that it oscillates around 0.80 gm. and we have obtained about the same results, while in man one finds between 0.80 and 1.20 gm. of glucose per liter of blood.

But, as we have said above, the quantity of sugar in blood varies with lactation and the food. In the course of lactation, Widmark and Carlens have shown that in apparently healthy cows in the same herd and on the same feed the percentage of glucose in the blood is always lowest in those giving the most milk. In the heifer and the dry cow it oscillates around 0.80 gm. per liter; in the milch cow it is about 0.60 gm. Our investigtaions have confirmed those of Widmark and Carlens but we have conducted them from another viewpoint and have attempted to verify if the establishment of lactation, after calving, would not bring about a marked decrease in the percentage of glycemia. Before parturition, always when milking is suspended, one finds the percentage of blood sugar raised; during the few days preceding parturition, in daily tests, we have found the figures vary from 0.90 gm. to 1.20 gm. per liter, with intermittent lactosuria, it is true, while after parturition the sugar in blood oscillates between 0.55 and 0.75 gm. We have made analogous observations on two goats, before and after parturition, finding 0.7 to 1.0 gm. before, and 0.55 to 0.65 gm. after parturition. To the time when lactation is established, if milking is regularly practised, one always observes a notable lowering of the percentage of glycemia.

Glycemia varies also with alimentation. When milch cows receive indigestible foods the percentage of sugar in the blood descends very low and near the point where hypoglycemia appears. Widmark and Carlens have observed, in milch cows nourished exclusively on the leaves of sugar beets, a glycemia equal to 0.40 gm. per liter, then to 0.30 gm., at which time one frequently observes disturbances due to hypoglycemia. When, on the contrary, milch cows receive concentrates, glycemia is increased but without ever attaining the percentage observed in heifers and dry cows, oscillating between 0.60 and 0.70 gm. per liter. The administration, then, of digestible food

must be a preventive treatment for milk fever; it permits the accumulation of important hepatic reserves which will be useful at the time the udder becomes active. The facts confirm this deduction, as we have already noted the statement of Maguire that English breeders feed pregnant cows concentrated food during the six weeks preceding parturition, thus avoiding milk fever and at the same time augmenting the yield in milk. One can at will, on the same animal, vary the glycemia by alternate feeding of concentrates and food less digestible.

One finds then, that in milch cows the percentage of glycemia is always lower than what is constant in dry cows and heifers and, according to the food given and the intensity of lactation, the amount of sugar in the blood can descend very low and arrive almost to the limit at which hypoglycemic disturbances appear, this condition manifesting itself very readily.

### The Hypoglycemic Syndrome in Milch Cows

*The signs of hypoglycemic syndrome in milch cows are identical with those of milk fever:* If our idea is correct and if milk fever is a manifestation of the hypoglycemic syndrome, one must be able to produce hypoglycemia artificially in a healthy animal and thus produce symptoms of milk fever. It is especially since the discovery of insulin by the Canadians that such an experiment has become possible. It follows clearly, from the experience of all investigators who have used insulin, that it is imperative, in order that hypoglycemic symptoms may appear, that the percentage of glycemia be lowered to a given level. In the rabbit the blood sugar must be no more than 0.45 gm. per liter in order that convulsions may appear; in man nervousness is noted at 0.70, some delirium at 0.40 or 0.50, convulsions at 0.30 gm.; Widmark and Carlens show that, in the bovine, glycemia must be lowered from 0.40 to 0.30 gm. and less for hypoglycemic symptoms to appear. As long as the percentage is not low, no alarming symptom is noted. There are evidently some individual variations in all species but the figures which we give are the average.

It must not be expected that giving one dose of insulin, proportionate to the weight of the subject, will bring promptly the hypoglycemic syndrome; numerous factors enter into play. One of the most important is the food. Although insulin must always be injected after a fast of from twelve to twenty-four hours, the previous food received plays an important part. This fact has

been observed by all authorities who have used insulin and recently Penau and Simonnet[21] have found that rabbits nourished with oats are much more resistant than those that are fed hay.

One must consider also individual variations. It has been observed in the rabbit that certain individuals of the same weight, same breed, sometimes of the same brood, resist longer the same dose of insulin. Banting, Best, Collip, Macleod and Noble[22] have reported the history of six rabbits in which they injected an equal quantity of the same insulin and each showed a different percentage of glycemia.

The method of administration must be considered. The intravenous injection always gives the most rapid results and at the same time the most marked. We used the intravenous method of administration.

Variations are equally due to age, the young being most susceptible. Certain breeds are less resistant. The action of insulin is increased in case of elevated temperature.

The condition of the organs, in particular the liver and kidneys, plays a certain part. The aged and sick appear more sensitive than the healthy subject. We shall have repeated occasion to insist on the importance of these factors.

Lactation, and this has not been mentioned before, plays a role of first importance. The milch cow is more sensitive than a beef cow and one injection of insulin partially dries up the milk secretion, thus a notable quantity of glucose which must be transformed into lactose is destroyed by insulin. That amounts probably to this, that the reserves in hydrate of carbon are minimized in animals in milk. Animals are most sensitive to insulin at the time after parturition when the milk secretion appears. In goats and dogs, uniformly nourished, we have given an injection of insulin before parturition and no trouble appeared, the lowering of the glycemia not having been sufficient; these same animals receiving two days after parturition the same dose of insulin, clear symptoms of hypoglycemia were shown. The injection of insulin, however, has but little effect on the composition of the milk or, and this is an interesting point, on the richness of lactose. Giusti and Rietti,[23] operating on goats, and Nitzescu and Nicolau,[24] experimenting on sheep, had identical results on this point; notwithstanding very strong doses of insulin, administered daily for ten consecutive days, and having caused at times a lowering of glycemia as much as 0.29 and 0.26 gm., they noted a very feeble diminution in lactose, as 0.46

gm. instead of 0.50 gm. to the liter, and some slight changes in the milk, as a small increase in the fat content and mineral phosphorus.

Repetition of the doses of insulin at short intervals, when the liver has no time to reconstitute its reserve, favors hypoglycemic syndromes.

With all these factors entering into play one cannot always reproduce in the cow the hypoglycemic syndrome by injecting insulin. We have made six tests on six cows; three times only did we note the trouble clearly, the glycemia being lowered to 0.30 gm., one case a semi-coma. In two cows, after a large dose of insulin, no trouble was observed, the lowering of the glycemia not having been sufficient. Widmark and Carlens have always noted a lowering of glycemia after injecting insulin, but this lowering has been variable with the subject and never proportional to the dose injected; it was on a subject in which a small dose of insulin was injected that the symptoms showed most clearly. One can, in order to explain these differences, make use of the facts which we have mentioned, and in particular the individual variations; but such variations are caused perhaps by organic lesions, such as the liver, endocrine glands, which intervene in the metabolism of carbohydrates, or to those of the vegetative nervous system. We recognize the existence of an unknown factor which possibly plays an important role in the syndrome of hypoglycemia. Even so, return to health is more or less rapid following an injection of glucose; sometimes even without the injection of glucose animals return to the normal state, glycemia improving spontaneously. We do not possess sufficient information on this subject because our experience has been limited to only six cows, due to the high cost of insulin; consequently we have used dogs principally. Most investigators in studying insulin have noted that on healthy animals the injection of glucose intravenously or subcutaneously is followed by the return to normal in a few minutes; however when the subjects are at the crisis it requires a little longer, from fifteen to twenty minutes; when they are large, more time still (it takes much longer to revive a large dog than a small one, according to Macleod).[25] We have observed the same thing, but many times our dogs have been a long time recovering; the convulsions produced by insulin will soon cease but the animal remains depressed from twenty-four to forty-eight hours. One of our experiment animals, an old dog, in spite of four

injections of ten gms. of glucose (two in the vein, one subcutaneously and one intraperitoneally), died in convulsions at the end of the fortieth hour; this dog eliminated the glucose purely and simply, as shown by our urinalysis, and in a carefully conducted autopsy we were able to find only lesions in the liver and kidneys; this was confirmed by a histological examination.

These statements perhaps explain why, if milk fever is a simple hypoglycemia, certain cows affected with this disease succumb in spite of udder insufflation or the injection of glucose. Maguire mentions[26] that in certain patients (and this is the experience of many practitioners) two or three insufflations of the udder or two or three injections of glucose must be made, for in practice he employs indifferently one treatment or the other, and some animals make a partial recovery after the treatment and a few succumb. In cows that have died of milk fever, Maguire finds the lesions of fatty degeneration of the liver and he thinks that the liver, thus affected, either is incapable of manufacturing glycogen, or of liberating glucose in sufficient quantity for the needs of the circulation. However it may be, the injection of insulin, when glycemia becomes sufficiently low in the cow, reproduces the classic symptoms of milk fever very completely; at least the comatose form, the most frequent, admitted by all, since in addition to the comatose form certain investigators actually describe a convulsive form and some raise the question of a special morbid entity under the name of eclampsia. Let us mention now that neither Widmark and Carlens nor ourselves have obtained in bovines, following the injection of insulin, the convulsive crises which are the rule in other species; it is true that the experiments have been limited, inasmuch as on six attempts we have but three times obtained a sufficient hypoglycemia to manifest symptoms; and Widmark and Carlens in seven attempts have obtained coma in but two instances.

When insulin is injected in a vein—we have used indifferently the jugular or a vein of the ear—the first symptoms begin to appear one hour after the injection. One observes a little agitation, restlessness, some localized trembling in certain groups of muscles, then a little later some apathy, dullness, and slight somnolency. At this time the cow lies down, generally refusing to arise in spite of urging. In one of our animals we observed, before decubitus, some difficulty in standing on the four feet; she staggered; when forced to move, the legs became crossed; stumbling increased, and the beast fell to the ground; being

particularly nervous, she attempted to arise but fell again. The decubitus is sternal at first, then somnolency increases, the cow seems unable to support the weight of her head and extends it upon the litter; lifting it for a moment and then replacing it upon the ground; frequently the head curves to one side of the body and remains in that position. One always notes a subnormal temperature, ½°, 1° C. or more. The respiration is slow and stertorous. There is paresis of the pharynx, some ptyalism, a little bloating, some paresis of the bladder and intestines with no micturation or defecation. These phenomena follow between the third and fifth hour. Soon the decubitis becomes lateral, the head is drawn around to the side and the cow presents the classic position of milk fever. We give two photographs of the same cow in hypoglycemic coma. All practitioners will recognize the characteristic position which they have so often seen in cows affected with milk fever. The symptoms of hypoglycemia appear when the glycemia descends to or below 0.40 gm. per liter. Till this percentage is reached one notes a little apathy and dullness. She lies down sometimes but can get up under the influence of excitement.

We will repeat that the dose to inject is very variable. In our first experiments we had some clear phenomena of hypoglycemic coma with 190 physiological units of insulin (Byle) in a cow weighing 350 Kg., in the fifth month of gestation, giving five to six Kg. of milk; we were not able to lower the glycemia sufficiently to obtain a clear syndrome with 600 physiological units in a dry cow weighing 430 Kg., nor with 290 physiological units in a dry cow weighing 280 Kg., nor with 490 physiological units in a cow weighing 450 Kg., in full lactation (sixteen Kg. of milk), nor with 390 physiological units in a dry cow weighing 340 Kg., etc. The cow photographed is five years old, gave eight liters of milk, and weighed 400 Kg. After twelve hours of fasting she received 600 physiological units of insulin (Byle) and manifested only a little somnolency. The fasting was prolonged and the next day 600 physiological units of insulin (Byle) were again injected. It was not until the second injection that the hypoglycemic syndrome showed itself in all its intensity, when the glycemia descended to 0.20 gm. to the liter.

Animals can be brought back to normal after a few minutes, following the injection of glucose, as, for example, our first cow, in which we did not allow the coma to continue for a long time. Sometimes animals come back to normal spontaneously; the last

cow used in our experiment (her photograph is shown) got up alone six hours after the injection. When the action of the insulin is exhausted, in fact, glycemia tends to return to normal. Even before the use of the Evers treatment the losses from milk fever were only fifty per cent; bleeding, which was then recommended, was truly a rational treatment, at least if the ideas that we hold are correct. R. Lepine, in fact, has demonstrated that a bleeding brings about a certain degree of hyperglycemia.

Most of our cows were able to get up alone; those of Widmark and Carlens remained in a state of coma up to the time of slaughter, sixteen hours after the injection, when the glycemia had raised to 0.45 gm. There, let us emphasize, was an obscure point in the action of insulin. Was it due to a difference in the product? They used a Danish insulin. Was it due to a difference in the state of health? They operated on an animal sick with aphthous fever and destined to be slaughtered. We do not know. What we can affirm at present is that the injection of insulin, when it lowers the glycemia sufficiently, will cause symptoms absolutely analogous to those of milk fever.

### Effects of the Evers Treatment in the Milch Cow

*Insufflation of udder brings about hyperglycemia:* Although the marvelous effects of insufflation of the udder are shown, it is not so in regard to its mode of action, and all pathogenetic theories brought forth for the past twenty years have failed to explain its efficacy. It was thought at first that the iodid of potash used neutralized the poison in the udder, but as this medicament may be replaced by such variable products as boiled water, air, oxygen, etc., it must be admitted that the substance injected acts in a purely mechanical manner, exclusive of any chemical action. Those partial to the circulatory theories believe that the tension exercised on the parenchyma of the udder, pressing back the blood of the udder toward other regions of the body, regulates the cerebral circulation or, perhaps, that the insufflation of air into the udder determines a marked augmentation of the arterial pressure, as was looked for in the Seitter demonstration. To those who believe in the auto-intoxication of mammary origin, the compression exercised on the vessels and on the glandular cells suspends the elaboration of toxic products and Delmer claims that insufflation causes an influx of polynuclear leucocytes which destroy the poisons. Hoyois believes that gas insufflation obstructs the veins and leaves to the arteries their permeability

and that there thus results a passive hyperemia susceptible of a local medicinal action similar to that which results from the Bier treatment. Widmark and Carlens and ourselves have investigated the action of mammary insufflation. Our conclusions are identical, viz., that insufflation always provokes an augmentation of sugar in the blood and that this augmentation is proportionate to the intensity of lactation.

Widmark and Carlens have conducted eight experiments on the cow and goat. In all they have noted the glycemia before insufflation, then after fifteen minutes, and continuing up to ninety minutes. They found that after the Evers treatment the glycemia, was progressively increased, attaining its maximum between the forty-fifth and sixtieth minutes. Before insufflation the glycemia was 0.80 gm.; in good milkers it may increase to 2.20 gms.; after sixty minutes it fell rapidly and continuously, reaching normal around the ninetieth minute. It is at this time that the regulatory mechanism of the blood is brought into play and the excess of sugar is eliminated by the kidneys. On some of the animals Widmark and Carlens investigated the nature of the sugar in the urine and have shown it to be in the form of glucose and lactose.

Our procedure differed: We made a few tests of the blood before and after insufflation but endeavored to collect all the urine to determine not only the quantity of sugar it contained but the nature of the sugar, glucose or lactose, by the ozozone method. Had we limited ourselves to determining merely the glycemia, the objection might have been made that hyperglycemia could be due to lactose and not to glucose. Milking was performed as completely as possible before insufflation of the udder, but a little milk remained in the acini. Insufflation, by the pressure which it produced, brought about the rapid absorption of the elements of the milk; and lactose in particular, as Porcher has shown, passed rapidly and in very large quantities into the blood. We made six tests, four on cows and two on goats. We will give here three results obtained on cows, in order to show the influence of the intensity of lactation on glycemia produced by insufflation; one experiment being on a cow nearly dry; another on one giving five liters of milk; the third in a fresh cow giving sixteen Kg. On the three cows insufflation was maintained five hours and all the urine collected for twelve hours. The results are shown in the accompanying tables.

TABLE I—*A thin, old cow, red and white; weight, 280 Kg; nearly dry (1 Kg. per day)*

| DAY | HOUR | QUANTITY OF URINE PASSED (GMS.) | QUANTITY OF SUGAR PER LITER (GMS.) | QUANTITY OF SUGAR PASSED (GMS.) | REMARKS |
|---|---|---|---|---|---|
| Jan. 6 | 10:00 a.m. | 900 | 2.75 | | Lactose |
| Jan. 6 | 10:30 a.m. | | | | Insufflation |
| Jan. 6 | 12:30 p.m. | 250 | 2.26 | 0.765 | Glucose (little and lactose (much) |
| Jan. 6 | 1:30 p.m. | 125 | 6.47 | 0.161 | Glucose (little) and lactose (much) |
| Jan. 6 | 3:30 p.m. | 80 | 3.4 | 0.272 | Glucose (little) and lactose (much) |
| Jan. 6 | 10:30 p.m. | 1270 | 6.47 | 8.216 | Glucose (little) and lactose (much) |
| Jan. 7 | 10:00 a.m. | | | | No sugar |

Total amount of sugar eliminated: 9.414 gms.

TABLE II—*Cow, twelve years old, black and white; weight 350 Kg., 5 months pregnant; giving 4 to 5 liters of milk per day*

| DAY | HOUR | QUANTITY OF URINE PASSED (GMS.) | QUANTITY OF SUGAR PER LITER (GMS.) | QUANTITY OF SUGAR PASSED (GMS.) | REMARKS |
|---|---|---|---|---|---|
| Jan. 13 | 10:00 a.m. | 1010 | | | No sugar |
| Jan. 13 | 10:30 a.m. | | | | Insufflation |
| Jan. 13 | 12:30 p.m. | 2400 | 3.31 | 10.94 | Lactose (much and glucose (little) |
| Jan. 13 | 1:30 p.m. | 400 | 30.90 | 12.38 | Lactose (much) and glucose (little) |
| Jan. 13 | 2:30 p.m. | 450 | 27.2 | 12.24 | Lactose (much) and glucose (little) |
| Jan. 13 | 3:30 p.m. | 250 | 24.72 | 6.18 | Lactose (much) and glucose (little) |
| Jan. 13 | 10:30 p.m. | 1500 | 11.82 | 17.73 | Lactose (much) and glucose (little) |
| Jan. 14 | 10:00 a.m. | | 5.44 | | Lactose (much) and glucose (little) |
| Jan. 14 | 10:00 p.m. | | 3.02 | | Lactose (much) and glucose (little) |
| Jan. 15 | 10:00 a.m. | | | | No sugar |

Total amount of sugar eliminated: 59.57 gms.

TABLE III—*Cow, ten years old, red and white; weight 450 Kg. Fresh, February 29; twins; one died during labor. Giving 15 Kg. of milk per day*

| DAY | HOUR | QUANTITY OF URINE PASSED | QUANTITY OF SUGAR PER LITER (GMS.) | QUANTITY OF SUGAR PASSED (GMS.) | REMARKS |
|---|---|---|---|---|---|
| Mar. 7 | 10:00 a.m. | | 0.90 | | Lactose |
| Mar. 7 | 10:30 a.m. | | | | Insufflation |
| Mar. 7 | 12:30 p.m. | 1300 gms. | 6.01 | 7.814 | Glucose (little) and lactose (much) |
| Mar. 7 | 1:30 p.m. | 450 gms. | 27.50 | 12.375 | Glucose (little) and lactose (much) |
| Mar. 7 | 2:30 p.m. | 470 gms. | 30.99 | 14.565 | Glucose (little) and lactose (much) |
| Mar. 7 | 3:30 p.m. | 350 gms. | 24.64 | 8.624 | Glucose (little) and lactose (much) |
| Mar. 7 | 10:30 p.m. | 5 liters | 11.92 | 59.60 | Glucose (little) and lactose (much) |

Total amount of sugar eliminated: 102.978 gms.

As may be seen, the quantity of sugar eliminated is proportional to the intensity of lactation; thus, 10 gms. for the dry cow, 60 gms. for the one giving five to six liters, and more than 100 gms. for the cow in full lactation.

We also took blood from the jugular and the mammary veins in order to determine the amount of sugar. Kaufmann and Magne,[27] in order to demonstrate the formation of lactose at the expense of glucose, have proved that a notable difference between the blood of the jugular vein and that of the mammary vein always exists when the udder is in full function; the udder consuming more sugar than the tissues of the head. We have made the same observations, but in determining the amount of sugar after insufflation we have seen the proportion not only become equal in the two veins but sometimes even greater in the mammary vein. This augmentation is due to the absorption of lactose.

It has been determined by the experiments of Widmark and Carlens, as well as our own, that insufflation of the udder provokes a marked hyperglycemia and that this hyperglycemia is proportionate to the intensity of lactation. As one can readily understand by examination of table II, this hyperglycemia follows occasionally some time later, twenty-four hours in general,

because the udder is not open immediately to the full capacity of air and the milk secretion is more or less lowered by this fact. In all our cases, the cows insufflated did not return to a normal milk flow for three or four days.

We believe that the Evers treatment acts altogether mechanically by annihilating the function of the udder; the liver continues to secrete the necessary glucose to function the udder and this glucose, not being utilized for the formation of lactose, accumulates in the blood and causes hyperglycemia. Mammary insufflation acts the same as ablation of the udder and provokes effects analogous to those which Porcher observed in his experiment on the origin of lactose. We believe it to be an exclusively mechanical action and that the nervous symptom does not intervene as a result of the mammary activity as might be supposed. It is known that massage of the udder by the calf and by milking provokes an abundant milk secretion and by *a priori* evidence it has been deduced that mammary insufflation will provoke hyperglycemia reflexly by massage of the udder. Experimentation contradicts this hypothesis. We have practiced mammary insufflation on dry goats, aged three years, and have determined the amount of sugar in the urine and glucose in the blood both before and after insufflation and have observed neither glycosuria nor hyperglycemia. We have also studied the action of the Evers treatment on blood pressure. This experiment is in operation at present; we will give the results at some future time.

In conclusion one can say that the Evers treatment, by suppressing mechanically the function of the udder, acts as ablation of the udder and provokes a hyperglycemia. This hyperglycemia is proportionate to the abundance of milk secretion.

### Hypoglycemia in the Course of Milk Fever

*Effects of injecting a solution of glucose:* If our ideas are correct it would appear at first glance that, in order to carry the last stone to the edifice and demonstrate milk fever to be a manifestation of the hypoglycemic syndrome, we have only to determine the amount of sugar present in the blood of cows suffering from milk fever to prove hypoglycemia and, further, to treat the disease successfully with fluid glucose. The problem, however, is complex, and in the first of these instances can be demonstrated only with difficulty. Maguire, in his interesting study, was able to cite two cases of milk fever where he had determined the

amount of sugar in the blood. In one he found at the time of coma, 0.40 gm. of sugar to the liter, and in the other 0.50 gm. After mammary insufflation, or the injection of glucose, the sugar was rapidly raised to normal and the cure followed at the same time. Our English confrère recalls a case where there had been little or no retention of milk; this case occurred because the lactosuria must at times fail in the course of milk fever, but such cases are relatively rare. It must not be forgotten that at the time when milk fever begins, the udder is generally filled with milk and, milking having been neglected, there is most often milk retention with consequent absorption of lactose into the blood at first and then into the urine. Porcher, in his studies on milk retention, insists on the fact, that, of all the component parts of milk, lactose is resorbed first and in the maximum quantity. In his studies on the urology of milk fever[28] he has recognized lactose fifteen times in fifteen cases and glucose six times in fifteen cases. (We will later explain the presence of glucose, mentioning now only the lactose.) This shows that in milk fever there is nearly always milk retention and lactosuria. Moreover, when testing the blood of an affected cow for the amount of sugar one almost always finds glucose and lactose from retention; the separation of these sugars and their amounts, simultaneously in the blood, is difficult. I know no practical method, except a true chemical analysis. We, ourselves, have not had an opportunity of procuring blood from an affected cow and of studying the glycemia; Professor Widmark's latest communication,[29] however, states that at the beginning of the disease he has found a glycemia equal to 0.1 or 0.2 gm. per liter. He also noted hyperglycemia at the end of two or three hours in cows in full coma before injecting glucose or applying mammary insufflation. He thinks with just reason that this hyperglycemia is due to lactose. The proof, however, is lacking by reason of difficulty in analysis, as the chemical method does not permit the separation and determination of the amount of glucose and lactose simultaneously present in the blood. All evaluations of sugar in blood being raised in animals affected with milk fever, and giving a normal glycemia or even hyperglycemia, would not invalidate the theory which we have supported. Our opinion having been given that lactosuria is seldom absent, hyperglycemia will usually be found if the differentiation in sugar is not made.

It might be expected that, when lactose has been injected intraperitoneally into an animal, all the lactose would be elimiated in and might be recovered from the urine. As a matter of fact, an experiment of Porcher and Tapermoux[30] demonstrated the contrary. They injected a definite quantity of lactose into the udder of a dry goat; they collected all the urine for twenty-four hours and then removed, by milking, all of the solution left in the udder at the end of that time and ascertained that of the total amount of lactose injected, one-quarter had been absorbed and utilized by the organism.

The effects of injecting a solution of glucose are already known, at least by a few investigators, the first of whom was the Canadian veterinarian (name unknown) reported by Neef to the Belgian Society of Veterinary Science. This practitioner cured a cow with milk fever by an injection of ten gms. of glucose. Maguire, in the *Veterinary Record*, claims that treatment by the injection of glucose, which he has employed recently in his practice, gives him as good results as the insufflation of air. Professor Widmark writes us that this method always gives perfect results. Certain of our confreres, in particular Mr. Vuillaume, of Lons-le-Saunier, and Mr. Bouchet, of Creil, have used this treatment on one case each, with success. Other veterinarians should try it and thereby confirm or nullify the theory of hypoglycemia.

*Regarding the question of the presence of sugar in urine:* Nocard[31] first mentioned this in 1885, then Saint Cyr and Violet,[32] Lucet,[33] and Albrecht,[34] but all of these investigators did not differentiate between sugar, glucose and lactose, found in urine, and this distinction has an important bearing. We must recall the fundamental work on this subject by Porcher, presented to the Central Society of Veterinary Medicine in 1902. He reported the results of urinalysis on fifteen cows affected with milk fever; in the fifteen cases he found lactose in more or less large quantities and justly remarked that the lactose was derived from milk retention; but, beyond the constant presence of lactose, Porcher recognized glucose in large quantities three times (see cases 2, 3 and 15, in his report) and three times in small quantities (see cases 4, 6 and 14). Cases 3, 4 and 15 furnish no information either as to the time when the urine was procured or the treatment that had been applied. For this reason it is not possible to discuss his findings. In case 2, reported to Mr. Porcher by our regretted confrère, Mr. Rey, of Lagnieu (Ain), one cow, on

October 31, 1902, showed the classical symptoms of milk fever; eight liters of urine were obtained by cathether; they contained lactose only; on November 1 and 2 the patient was much better and glucose appeared in the urine. The report is absolutely mute on the subject of treatment; it is probable that Mr. Rey used the Evers or Schmidt treatment (in use then) and that this treatment had caused an augmentation of glycemia which, at first normal, had gone beyond the threshold and caused the glycosuria recognized by Porcher. Cases 4 and 6 were reported by Mr. Parent, a veterinarian of Malesherbes (Loiret). In the first-mentioned the patient showed the disease at five o'clock in the morning; mammary insufflation was applied; urine was collected at nine-thirty a.m. and found to contain a small quantity of lactose and an infinitesimal quantity of glucose. In the second, the patient became sick December 19, 1902; the urine secreted this day contained only lactose; the form of treatment on this date is not mentioned but the next day, December 20, Mr. Parent applied the Schmidt treatment, the cow got up and the urine contained a little glucose. It is true that in all these cases glucose did not appear in the urine until after the Evers or Schmidt treatment. These latter cases produced a hyperglycemia which passed the limit and caused the glycosuria which was observed.

### Theory of Hypoglycemia

It would appear to be true, following the arguments that we have set forth, that milk fever must be due to a hypoglycemia; by what is it caused? Maguire attributes it to disturbance of the endocrine glands. In the course of gestation, he says, adrenalin is elaborated in large quantity, which causes, in a pregnant cow, a free glycosuria and an increase in blood pressure. This adrenalin, secreted in too great abundance, causes an exhaustion of the suprarenal capsules. Insulin, under an unknown influence, is secreted in abundance after parturition; it cannot be neutralized in sufficient quantities by adrenalin and the excess of insulin causes a diminution of blood pressure and an abundant hydration of the blood, with, as a consequence, the precipitation of albuminoid particles in the cerebral capillaries, provoking all the symptoms of milk fever, as fall of blood pressure, coma and death. The injection of air or oxygen into the udder, or, better, the intravenous or subcutaneous injection of a solution of glu-

cose, will cause the fragments of albuminoid particles to be precipitated and bring about a cure.

In reviewing milk fever it clearly appears that this disease is caused by intense functioning of the mammary gland. We have tried to demonstrate that at the time of parturition, when the udder begins to function, it produces a marked diminution of glycemia and it appears preferable to admit, since we know from the time of Porcher's experiments that lactose is elaborated at the expense of glucose in the blood, that it is the disbursement started by milking which causes hypoglycemia. If the demand of the udder is great, as is the case in heavy milkers, glucose is used in such quantity that it can cause a marked lowering of the percentage of sugar in the blood. Hypoglycemia and its train of consequences follows. In animals selected with a view to milk production, milk fever appears to be the result of a momentary want of coordination between the udder in full function and the organs charged with furnishing to the blood the constituents of milk. More precisely speaking, a want of coordination between the organs which intervene in the metabolism of carbohydrates and the udder. The injection of glucose and mammary insufflation—the first by reestablishing the percentage of glucose in the blood and the second by annihilating the functioning of the udder, thus permitting the blood to recuperate its tenure of glucose—reestablishes this coordination necessary to life. We can recall the calculation of Widmark and Carlens, which states that a cow giving twenty-eight Kg. of milk renews all the glucose in the blood every fifteen minutes.

Under the pressure of specialization it is not only for the constituents of milk that the organism cries aloud in animals amenable to organic disease. In heavy milkers, notwithstanding intensive feeding of a ration perfectly calculated and balanced, American cattle-breeders have proved the appearance of diseases of the skeleton, with demineralization of bone, together with spontaneous fractures from this cause. The elaboration of milk demands a large amount of lime; that of alimentation is not sufficient; bone formation is altered, and increasing the proportion of lime in the ration does not always cause the disappearance of these troubles. But little is known about calcium metabolism. It is understood that the endocrine glands (parathyroid, thymus, thyroid and genital glands) intervene. Are there lesions of these glands affecting a change of the vegetative nervous system in the coordination of their actions? We do not know. We do

know that for the regulation of sugar there is intervention of the liver, pancreas, suprarenals, kidneys, hypophysis, thyroid bodies and even the spleen and salivary glands, and that the action of these glands is coordinated by the nervous system. Furthermore, beside the momentary lack of coordination most often noticed and which can be caused by specialization in milk production, a lesion in one or more of these glands can provoke, at the time when milk secretion is on the decline, a trouble. The cows which either recover slowly or succumb to milk fever, in spite of the Evers treatment, have, without doubt, lesions of the organs which regulate the metabolism of sugar; lesions which perhaps were only partially recognized. Cagny and Delamarre, Ehrhardt, and Maguire have already mentioned a fatty degeneration of the liver. The same may be true of cows not cured of milk fever and of certain animals which, having received insulin, are not cured by the injection of glucose. There may be two forms of milk fever: one, due to a lack of momentary coordination between the udder and the glands which regulate the metabolism of carbohydrates, relatively benign if rational treatment is used, and a second form, accompanied by organic lesions. This latter is always grave and responds badly to the usual treatment. One sees by this that the question of the nature of milk fever is not yet completely elucidated and that we are far from having solved the question. The theory of hypoglycemia explains clearly, in all instances, the cases of milk fever appearing before parturition. One knows that milk secretion begins before parturition, at least to some extent, in heavy milkers.

Upon consideration of the facts known up to the present, a new treatment for milk fever can be extolled. It is the injection of a solution of glucose. This may be given intravenously or subcutaneously. The first method is the more rapid. Maguire injects 200 gms. of a ten per cent solution of pure glucose. We recommend the same quantity of a twenty per cent solution. This injection is without danger because the excess of glucose is eliminated in the urine. The revival of animals is produced at the end of a variable time, as in insufflation, but in general a little sooner, or in from one to two hours. The treatment is simple and more professional than insufflation. Practitioners should verify its efficiency.

The preventive treatment of English cattle breeders is also worthy of recommendation. This treatment, which they call

"steaming"—a word which can be translated into French as "put under pressure"—consists of feeding animals, six weeks before calving, digestible food of constricted volume. The following is an example of steaming, by Maguire:

| | |
|---|---|
| Oil cake of shucked nuts | 1 part |
| Oil cake of soja | 1 part |
| Oil cake of rice | 4 parts |
| Oil cake of palm | 1 part |
| Corn gluten | 1½ part |
| Bran | 1 part |
| Molasses | 1 part |

This ration consists of six per cent fat and twenty per cent albuminoids. In using it Maguire holds that the milk production will be increased from ten to twenty-five per cent and that milk fever will disappear. On some milking herds of Shorthorns this method of feeding was used and Maguire saw the percentage of milk fever cases fall from six-tenths of one per cent to none. The truth is that this treatment acts by augmenting the reserves of the organism in *albuminoid* material, carbohydrates and fats and of these, as phsyiologists have proved, the two former are transformed into sugar.

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## KANSAS MEETING

Secretary Bower has announced that the twenty-third annual meeting of the Kansas Veterinary Medical Association will convene at the Jayhawk Hotel, Topeka, Kans., January 12-13. The committees having the preparation of the program in charge have worked hard and untiringly to make the convention the best one ever held. Commissioner Mercer will contribute to the program in the form of an address entitled "The Relation of the Veterinarian to the Live Stock Sanitary Commissioner's Office." Among the veterinarians from out of the State who will contribute to the program are Dr. Ashe Lockhart, of Kansas City, Mo., who will discuss "Common Diseases of Sheep"; Dr. J. V. Lacroix, of Evanston, Ill., who is down for an extemporaneous talk; Dr. R. C. Moore, of St. Joseph, Mo., who will give an illustrated lecture on "Surgical Diseases of the Udder"; and Dr. Sivert Eriksen, of Kansas City, Mo., who will demonstrate a practical test for bacillary white diarrhea. Many well-known Kansas practitioners will contribute to the program.

## O. S. U. RADIO PROGRAM

As promised in the November issue of the JOURNAL, we are giving the list of radio addresses to be delivered by the members of the faculty of the College of Veterinary Medicine, Ohio State University, during the months of January and February. These addresses will be broadcast by station WEAO, Wednesday evenings, at 8:00 p. m.

*January 19.* "The Care of Animals," by Dr. J. H. Snook.

*January 26.* "What Your Uncle Sam is Doing to Prevent Animal Diseases," by Dr. J. D. Grossman.

*February 2.* "What Your State Should Do to Control Animal Disease," by Dr. Walter R. Hobbs.

*February 9.* "What Your City Should Do to Protect Your Health," by Dr. J. H. Rietz.

*February 16.* "Why Vaccinate?" by Dr. R. E. Rebrassier.

*February 23.* "Veterinary Education," by Dr. D. S. White.

# OBSERVATIONS ON THE DURATION OF CHOLERA IMMUNITY IN BABY PIGS FOLLOWING SERUM AND VIRUS TREATMENT*

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The possibility of further reducing the cost of immunizing swine against cholera, by administering serum and virus to pigs before they are weaned, has been suggested in the results of investigations conducted by Dorset and Niles. Practicing veterinarians in treating young pigs under field conditions have reported results that were encouraging and some that were discouraging. It must be acknowledged, therefore, that susceptibility to cholera, though relatively rare in pigs treated as babies or in pigs treated at any age, may nevertheless occur. Such irregularities, however, cannot be regarded as evidence that baby pigs are any less subject to permanent immunization than pigs that have been weaned.

A résumé of the experimental data suggests that healthy pigs of any age can be rendered permanently immune, or at least relatively so, by the injection of potent anti-hog cholera serum and virus. A majority of the baby pigs treated in our experiments, over a period of four years, have proved immune to cholera at market age, yet in two groups, treated in 1923 and 1924 respectively, a small percentage proved susceptible to cholera when exposed at market time. The cholera susceptibility in these pigs, in so far as we have been able to determine, while not related to the age at which the pigs were vaccinated, is probably associated with some specific cause or causes the evaluation of which is an important problem confronting the veterinarian and breeder.

It is not the purpose of this paper to attempt an explanation of the occasional cholera susceptibility reported in other investigations, but to discuss the results of our own experiments wherein this difficulty was encountered. In this discussion it is not presumed that a definite explanation of cholera susceptibility follow-

*Presented at the sixty-third annual meeting of the American Veterinary Medical Association, Lexington, Kentucky, August 17-20, 1926.
†Resigned.

ing apparent immunity can be presented, but some of the factors entering into this complex problem may be mentioned with the view to stimulating further observations along this line. The expiration of cholera immunity in baby pigs following the serum and virus treatment was encountered in pure-bred and grade herds at the Harris farms in Central Illinois, in 1923; and in the pure-bred Experiment Station herd, in 1924.

### Harris Farm Pigs

In the spring of 1923 a contract between the Board of Trustees of the University of Illinois and the Harris Farms of Champaign County, Illinois, provided that the pigs on five of their farms would be placed under the jurisdiction of the Laboratory of Animal Pathology and Hygiene for the specific purpose of studying the practicability of administering serum and virus before weaning. Between February 27 and April 11, 1923, 112 pure-bred and grade sows farrowed 741 pigs that were used in the experiments. Each litter was permanently marked at birth, with notches in the ears, for future identification. Dr. D. L. Cecil, the attending local veterinarian, and Mr. James Dowell, manager of the farms, supervised the marking of the pigs. Records of all animals were filed at the University as soon as the identification was complete.

An average of 6.5 pigs per litter was farrowed. During the time elapsing from farrowing to immunization, an average of less than one pig in a litter died from injury and non-specific causes. Six hundred forty-three pigs, or 86.77 per cent of the 741 pigs farrowed, were given serum and virus by Dr. Cecil on April 24, 1923. The pigs at the time of treatment varied from 2 to 8 weeks of age. Two hundred sixty-nine were 7 to 8 weeks old, 181 were 6 to 7 weeks old, 115 were 5 to 6 weeks old, 50 were 4 to 5 weeks old, 5 were 3 to 4 weeks old, and 23 were 2 to 3 weeks old. (See table I.) The amount of serum for each pig varied between 10 and 30 cc, while the average dose was 24.9 cc. The average dose of virus was 2.09 cc. At the time of treatment 2 pigs received virus alone. Both developed symptoms of cholera and died.

### Loss from Parasitism

Following the administration of serum and virus, weekly inspections of the pigs were made. For a period of six weeks no evidence of illness other than transitory stiffness, observed immediately following inoculation, was detected. During the latter

half of June, July, and August, many of the animals showed symptoms of unthriftiness and several of them died. The syndrome did not resemble cholera, and the affected pigs at autopsy showed no lesions of this disease. Several visibly sick animals were destroyed for autopsy at the different farms, while typically affected pigs were delivered to the laboratory for study. Autopsies revealed unmistakable evidence of a severe infestation of *Ascaris lumbricoides* and *Macracanthorhynchus hirudinaceus*, as well as lesions of colitis. The gradual development of unthriftiness and

TABLE I—*Cholera exposure at market age of pigs immunized as babies (Harris farms)*

| PIGS IMMUNIZED MARCH 24, 1923 | | PIGS DIED FROM INTESTINAL PARASITES AND NECROTIC INFECTIONS[1] | | EXPOSED TO CHOLERA AT MARKET AGE[2] | | |
|---|---|---|---|---|---|---|
| AGE (Weeks) | NUMBER | NUMBER | PERCENTAGE | EXPOSED | DIED | |
| | | | | | NUMBER | PERCENTAGE |
| 7-8 | 269 | 77 | 28.6 | 168 | 11 | 6.54 |
| 6-7 | 181 | 80 | 44.2 | 75 | 12 | 16.00 |
| 5-6 | 115 | 56 | 48.7 | 35 | 8 | 22.85 |
| 4-5 | 50 | 26 | 52.0 | 18 | 5 | 27.77 |
| 3-4 | 5 | 0 | — | 4 | 0 | 0.00 |
| 2-3 | 23 | 14 | 60.9 | 4 | 0 | 0.00 |
| Totals | 643 | 253 | 39.34 | 304[3] | 36 | 11.84 |

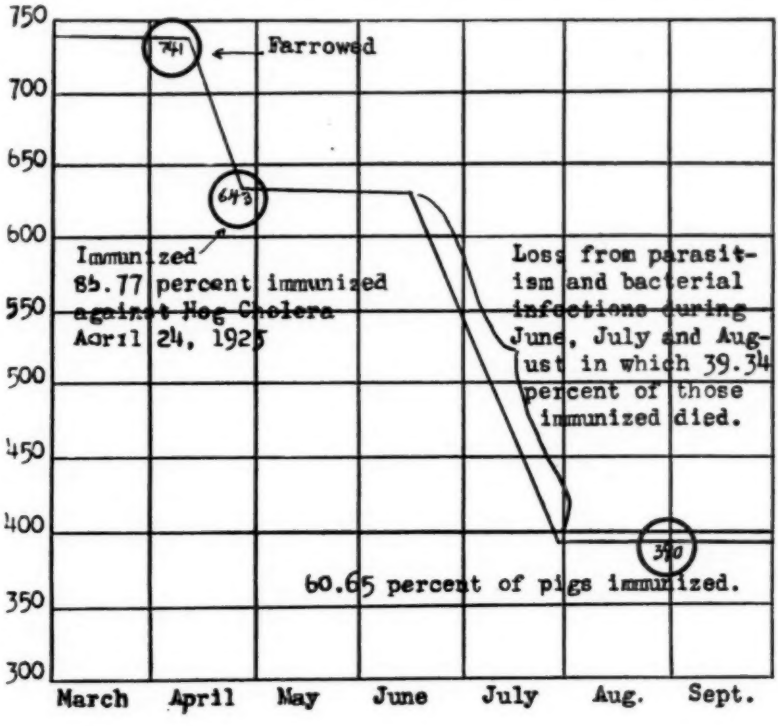
[1]May, June, July, 1923.
[2]October, November, December, 1923.
[3]Eighty-six of the immunized pigs were not used for cholera exposure.

the chronic termination of the parasitic and bacterial infections were recognized as serious, yet a full realization of the damage was not appreciated until the pigs were assembled for cholera exposure late in the fall. A summary of the losses in the five herds, according to the age of the pigs at the time of treatment, is given in table I. It is noted that the heaviest losses occurred in pigs that were farrowed late and consequently were the youngest at the time of treatment, and that these losses occurred irrespective of the farm on which the pigs were raised. A total of 253 pigs, or 39.34 per cent of the 643, succumbed from causes

other than cholera, presumably from intestinal parasites and secondary bacterial infections. (See table II.)

The mortality of each group, according to age at the time of treatment, is included to complete the history of the animals exposed to cholera at market age. During the summer months the following pigs died from parasitism and intercurrent bacterial infections. Seventy-seven of 269 pigs immunized at 7 to 8 weeks of age, or 28.62 per cent; 80 of 181 immunized at 6 to 7 weeks of age, or 44.2 per cent; 56 of 115 immunized at 5 to 6

TABLE II—*Losses in pigs from parasitism and secondary bacterial diseases*




weeks of age, or 48.7 per cent; 26 of 50 immunized at 4 to 5 weeks of age, or 52.0 per cent; and 14 of 23 immunized at 2 to 3 weeks of age, or 60.9 per cent. (See table I.)

## EXPOSURE TO CHOLERA

Beginning in August and continuing through September, October and November, losses subsided and the health and vigor of the herds improved. The gain in weight made during this time was quite satisfactory; 86 animals, however, were not used in the exposure tests. As the healthy hogs approached market

age, 310 were moved one to two miles in three groups of 100, 106, and 104, for cholera exposure. Two groups were exposed October 29 and November 16, respectively, by administering to each animal 5 cc of hog cholera virus. On December 13, 1923, the third group was exposed in cholera-infected pens. No difference was observed in the infectious character of hog cholera virus and pen exposure in the rate of susceptibility encountered in the three groups. Following exposure all animals were held three weeks before conclusions were finally drawn as to their resistance to the virus.

### Respiratory Infection

At the time the pigs were exposed to cholera, the weather was cold and somewhat disagreeable. A few animals on arrival at the isolation quarters showed suggestive symptoms of a respiratory disease. The symptoms consisted of inappetence, slight sluggishness, and abdominal respiration. Those showing the slightest clinical symptoms or a temperature of 104° F. were rejected in the first two groups. This included two pigs in the first group and four pigs in the second group. These animals in isolation made an uneventful recovery. The third group, which included 104 animals, was rested for five days following delivery to isolation quarters for cholera exposure before their temperatures were taken. In this group 32 animals were found with temperatures of 104° F. and over. The clinical manifestations in the affected animals were regarded as transitory in view of the fact that the six rejected pigs in groups 1 and 2 recovered. In the third group the relatively large number showing borderline temperatures suggested the advisability of limiting the cholera exposure to contact with infected pens, notwithstanding the fact that clinically not more than six animals seemed affected.

### Results of Exposure to Cholera

The results of exposing the 304 hogs to cholera may be briefly summarized, on the basis of age at time of treatment and age at time of exposure, as follows: Of 168 hogs immunized at 7 to 8 weeks of age and exposed to cholera at 7 months, 23 days, to 9 months, 16 days, 11 or 6.54 per cent succumbed. The variation in age at time of exposure is accounted for by the three different dates at which exposure was made. Seventy-five hogs of a group of 181 pigs immunized when 6 to 7 weeks old and exposed to cholera at 7 months and 16 days to 9 months and

5 days, suffered a loss of 12, or 16 per cent. Thirty-five hogs of a group of 115 immunized when 5 to 6 weeks old and exposed at ages of 7 months and 9 days to 8 months and 28 days, suffered a loss of 8 animals, or 22.85 per cent. Eighteen hogs of a group of 50, immunized when 4 to 5 weeks old and exposed at 7 months and 2 days to 8 months and 21 days, suffered a loss of 5, or 27.77 per cent. Four hogs of a group of 5, immunized at 3 to 4 weeks of age and exposed at 6 months, 26 days, to 8 months, 14 days, as well as 4 hogs of a group of 23 immunized at 2 to 3 weeks old and exposed at 6 months, 19 days, to 8 month, 8 days, proved resistant to cholera. (See table I.)

Of the 304 hogs given serum and virus between 2 and 8 weeks of age and exposed to cholera at market age, 36, or 11.84 per cent, proved susceptible to cholera. The animals died within 10 to 16 days after exposure, showing symptoms suggestive of cholera. The autopsy findings were also suggestive of cholera, while the filtered blood from fatally affected animals produced symptoms of cholera and death in a susceptible pig. To establish further the character of the infection, four cholera-susceptible pigs were delivered to the farm and placed in the infected quarters. Two received serum and virus before entering the pens, while the other two were left untreated. The untreated pigs died, showing symptoms and gross pathologic lesions indicative of cholera, while the serum-and-virus-treated pigs remained healthy.

The mortality from cholera observed in the different groups at market age yields very little, if any, definite evidence to suggest that the cholera susceptibility observed was related to the age of the pig at the time of inoculation. The possible significance of the respiratory infection observed in the animals at the time of exposure to cholera is not so easily eliminated as a factor even though animals showing clinical symptoms and fever in the first two groups were carefully discarded.

The suggestive correlation between the rate of cholera susceptibility and the loss sustained from intestinal parasitism and necrotic intestinal infections in the different groups is a matter of interest, but more supporting data are necessary before conclusions can be drawn. Parasitism and other conditions responsible for lowered vitality are probably not excluded as possible factors exerting an influence on the stability of cholera immunity. It must be admitted that such factors are difficult to measure in a precise manner.

### Experiment Station Pigs

In conducting a comparable experiment in pigs at the Experiment Station Farm, in 1924, the importance of the health of the immunized hogs in resisting cholera rather than the age of the animal at the time of the simultaneous treatment was again suggested. The pigs in the 1924 tests were ear-marked when farrowed. Two hundred sixty pigs received anti-hog cholera serum and virus on May 13, 1924. The pigs varied from 2 to 12 weeks of age at the time of treatment. (See table III.)

Table III—*Cholera exposure at market age of pigs immunized as babies (Experiment Station herd)*

| Pigs Immunized May 13, 1924 | | Exposed to Cholera Four Months After Immunization | | Exposed to Cholera Five Months After Immunization | | |
|---|---|---|---|---|---|---|
| | | | | | Died | |
| Age (Weeks) | Number | Number | Results | Exposed | Number | Percentage |
| 10-12 | 24 | 5 | Remained healthy | 9 | 1 | 11.11 |
| 8-10 | 95 | 34 | Remained healthy | 29 | 1 | 3.44 |
| 7-9 | 59 | 10 | Remained healthy | 25 | 2 | 8.00 |
| 6-7 | 12 | 3 | Remained healthy | 7 | 1 | 14.28 |
| 2-5 | 70 | 13 | Remained healthy | 50 | 8 | 16.00 |
| Totals | 260[1] | 65 | | 120 | 13 | 10.83 |

[1]Seventy-five of the immunized pigs were not exposed to cholera at market time.

The dose of anti-hog cholera serum varied from 10 to 30 cc, with an average of 25 cc. Three cc of virus were given each pig. Frequent visits to inspect the pigs were made. Losses following vaccination from intercurrent infections and parasitism were negligible, and served as a distinct contrast to the experience in the experiment of the preceding year on the Harris farms. On September 11, 1924, 65 of the pigs from different litters, varying from 4 months and 2 weeks to 6 months and 3 weeks of age, were given subcutaneous injections of 5 cc of cholera virus. The pigs in this group at time of immunization varied in age

from 2 to 12 weeks. Five of the pigs were immunized at 10 to 12 weeks, 34 at 9 to 10 weeks, 10 at 7 to 9 weeks, 3 at 6 to 7 weeks and 13 at 2 to 5 weeks. All pigs of this group exposed to cholera on September 11, 1924, remained healthy. (See table III.)

One hundred twenty pigs from the same litters were exposed to cholera in a similar manner on October 9, 1924. The animals varied in age from 5 months and 13 days to 7 months and 28 days, including 9 pigs immunized when 10 to 12 weeks old, 29 pigs immunized at 9 to 10 weeks, 25 pigs immunized at 7 to 9 weeks, 7 pigs immunized at 6 to 7 weeks, and 50 pigs that were immunized at 2 to 5 weeks.

Two days following the administration of virus, many of the animals in this group became suddenly ill. Their appetites were indifferent, and the animals lost rapidly in weight. Affected animals showed dyspnea and elevation of body temperature. In a majority of the animals the symptoms subsided after the third day, though other animals continued weak and fatigued. Similar symptoms were also manifest in animals of the herd not included in this experiment; the mortality among those animals was negligible, while 13, or 10.83 per cent, of the cholera-exposed group succumbed. Autopsy lesions and inoculation of pigs with filtered blood-serum from infected animals gave evidence of cholera. (See table III.)

## Summary

Observations on the duration of immunity in two groups of pigs, including 304 animals in 1923 and 185 in 1924, that were immunized to cholera as babies and exposed at market age, show that approximately 90 per cent retained immunity to cholera. Other pigs immunized as babies in similar experiments not reported in this paper have consistently proved immune to cholera at market age. The mortality following exposure to cholera at market age, in our opinion, is such as to support the belief that cholera immunity probably is not dependent on the age of the animal at the time of inoculation, notwithstanding the fact that a small percentage of the baby pigs immunized while sucking proved susceptible to cholera at market time.

In the 1923 group the presence of a mild respiratory infection was observed previous to cholera exposure. The illness was transitory in animals that were not exposed to cholera. A similar disease appeared two days following virus injection in the 1924 group. An effort was made to eliminate all animals

showing any clinical evidence of ill health preceding cholera exposure in 1924, yet 36 of 304 hogs, or 11.84 per cent, died of cholera. The filtrable virus of cholera in fatal cases was demonstrated by inoculation of susceptible pigs.

Somewhat similar results were observed in the 1924 group of 185 pigs. These pigs were immunized as babies and exposed to cholera in two separate groups by administering 5 cc of hog cholera virus. In the first group, which included 65 animals, all proved resistant to cholera. Approximately one month later, 120 of the pigs were exposed in a similar way, and 13 or 10.8 per cent died. On the second day following cholera exposure, a respiratory infection suddenly appeared in the herd. It is believed that this disease was in no way related to the administration of the virus.

In view of the fact that other groups of pigs treated with serum and virus as babies, not reported in this paper, have proved immune to cholera at market age, the part played by respiratory infections at the time of cholera exposure is suggested. Furthermore it is surmised that respiratory infections in the incubative stages in swine may escape the attention of the clinician. If this conjecture be true, the relation of secondary bacterial infections, as well as other factors, may be operative in pigs irrespective of their age at time of treatment. The correlation between the rate of loss from *Ascaris lumbricoides* and *Macracanthorhynchus hirudinaceus* and cholera susceptibility is of interest, though it is not sufficiently definite to warrant any conclusion. The possibility of parasitism lowering the vitality and influencing the strength and length of the immunity, however, should not be entirely disregarded.

Cholera susceptibility in pigs following immunization is the exception, irrespective of age, but it must be conceded that it has been observed occasionally for many years. Until more definite and specific information is obtained to explain the cause or causes of occasional susceptibility in serum-virus-treated pigs, it is important that veterinarians assist swine-breeders by correcting the widespread impression that pigs given serum and virus will never contract cholera. Likewise, it seems important that we recognize that baby pigs can be immunized by administering serum and virus. This slight modification of our past teaching policy, together with recognition of the possible importance of diseases other than cholera with their influence on the stability of cholera immunity, may tend

to guard and better fortify the interests of the breeders in controlling hog cholera in animals previously immunized. In conclusion, it does not seem that the practice of immunizing baby pigs should be recommended as a procedure to replace immunization following weaning, but that the method might be tried in herds with the approval of the owner without incurring unusual risk.

### Discussion

Chairman Cahill: No doubt the paper just read by Dr. Tunnicliff appeals to me more than it does to anybody else.

There has, for several years, been a marked discrepancy between the findings obtained in my studies of this problem and those of some other workers. My investigations indicated that baby pigs could not be satisfactorily immunized in a sufficiently large percentage of the cases to make this a satisfactory procedure. Since there can be no question of the sincerity and honesty of purpose of each school, I have for some time been of the opinion, as pointed out in a previous paper, that these different results could be ascribed only to a difference in the conditions under which the experiment animals were maintained. The findings presented in this paper serve to strengthen such a belief and it is to be hoped that much more work, particularly as it applies to field conditions, will be done in order that this really great problem may be solved.

Dr. A. T. Kinsley: Dr. Tunnicliff's paper has been very interesting and because of the limited time only a few of the important points will be mentioned.

First: Pigs may die of other diseases than cholera.

Second: The effect of other diseases and depressing influences in diminishing resistance or overcoming cholera immunity should be given consideration.

Third: Baby pigs can be immunized against cholera, according to the reports of Cahill and Dorset, as well as the report we have just heard. However, the losses from cholera later in life are too large for this procedure to be recommended and carried out by practitioners.

Dr. J. W. Benner: I am very much interested in this subject and have been experimenting on it since 1922. At the present time it seems to me that a great deal more work should be done.

A most important point brought out in the paper is that practitioners should make careful examinations of swine herds and their environments and let the conditions found guide in choosing a method of vaccination against cholera.

If Dr. Cahill had condinued his pig immunity experiments I am of the opinion that his attention would have been directed to factors other than age that influence permanency of immunity.

I wish to take time to speak of only one of the factors which I consider of the utmost importance in producing a solid and lasting immunity in baby pigs. This factor is the cholera virus used in the simultaneous treatment. In 1922 and 1923, pigs vaccinated by the simultaneous method when three weeks old, and exposed by injecting 2 cc of virus when they were six or seven months of age, proved to be immune to that dose of virus.

In 1924 more pigs were treated when three weeks old by the simultaneous method. That fall, six or seven months from the time of vaccination, an exposure to virus was given by hyperimmunizing the animals, in which process 5 cc of virus per pound live weight was injected intravenously. Some of the animals died. I then dropped back to an exposure with 2 cc of virus to test the immunity of the remainder of the herd and had no further losses. This showed a grade of immunity to be present that would protect against 2 cc of virus injected intramuscularly but not sufficiently high to protect against 5 cc per pound live weight injected intravenously.

I also found, in 1924, that the virus I had used to immunize pigs three weeks old, and which failed to produce the highest grade of immunity, likewise failed to produce good hyperimmunes. Two series of our serum made in 1924

could not be used on account of low potency. We can logically expect that a virus that will not produce a good immune will also fail to produce a good hyperimmune and that is exactly what happened.

Dr. Dorset told me that he had a similar experience in his pig immunity in 1924 but I believe he did not mention the hyperimmunizing power of the virus he used. Dr. Dorset suggested to me that the age of pigs from which virus is drawn may have an influence on its potency. I have evidence that such was the case with the virus I used on baby pigs in the spring of 1924. I used some susceptible shotes that were five to six months old, weighing 100 pounds or more. Shotes of this age and size frequently do not react so sharply to an injection of virus. Their temperatures, instead of going up to 106° and 107° on the fifth and sixth days, will stay around 104.5° and 105.5° and instead of being ready to kill by the seventh day they are ready about the tenth. Virus from such shotes is capable of producing visible sickness and typical lesions of cholera but it is apt to produce a low grade of immunity when used in the simultaneous treatment and a low grade of hyperimmunity when used in serum production. Acute cholera is absolutely necessary to produce a highly potent virus and the fresher the virus can be used, whether to produce more virus or in the simultaneous treatment, the better. We should direct our attention to the age of the donor of the virus, as well as to the recipient of it, and also to the age of the virus.

There are other factors which may influence the potency of virus and many more that may influence the conferring of a solid and lasting immunity, but I will not take more of your time.

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## RESEARCH ON SURGICAL SUPPLIES

According to Dr. E. R. Weidlein, Director, Mellon Institute of Industrial Research, University of Pittsburgh, the firm of Johnson & Johnson, manufacturer of surgical supplies, New Brunswick, N. J., has established at the Institute a fellowship that will study the exact requirements of surgeons and other medical specialists in the way of sundries, with the joint aim of developing new supplies that are needed and of standardizing the products now in use. An investigation will also be made of the processes of renovating used supplies, and several other industrial fellowships of the Institute will cooperate in devising satisfactory procedures.

Dr. Frederic H. Slayton will be in direct charge of this comprehensive research. The fellowship will be operated in a totally unbiased and independent manner, in accordance with the Mellon Institute system, and all its investigations will be conducted primarily for the benefit of the public. It is the plan to report the results in appropriate periodicals, as the various phases of the studies are concluded.

In carrying on this work, Dr. Slayton and the Institute's executive staff invite the concurrence of all interested organizations. They are especially desirous of securing the close collaboration of hospital executives and of members of the medical and allied professions.

# RESULTS OF INJECTING PREGNANT HEIFERS WITH *BRUCELLA ABORTUS* ISOLATED FROM MAN*

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During the last two years there have been described in the United States, as well as in other countries, typical cases of undulant fever in man from which *Brucella abortus* has been isolated rather than *Brucella melitensis*. Although these two microorganisms are very similar in their biological characteristics, Evans[1] has shown that they can be differentiated with certainty by the agglutinin-absorption test. Her findings have been verified by Meyer and Shaw.[2] Other workers have described various characters and methods which might differentiate these two organisms, but further observations have shown that they cannot always be relied upon.

The author has had an opportunity to study ten cases of undulant fever in man due to a microorganism indistinguishable from certain strains of *Brucella abortus*, which have been isolated from porcine and bovine sources. The cultures isolated from the blood of man have been recovered in three different ways: by sealing the tubes with sealing wax, by replacing 15 per cent of the atmosphere in which they were grown with 10 per cent $CO_2$, and by guinea pig injection. Positive identification of the cultures has been accomplished by the study of the agglutinins in the antiserum, the cultural characteristics, the lesions produced by them in guinea pigs and their effect upon pregnant heifers injected intravenously.

Abortion has been produced experimentally in pregnant, susceptible cows and heifers when they have been injected intravenously with various suspensions of *Brucella abortus* of bovine and porcine origin. Invariably abortion has been produced by this method when bovine strains have been used. According to Cotton,[3] however, *Brucella abortus* of porcine origin does not regularly produce abortion in cattle.

Five heifers in their first pregnancy were purchased from herds, where, clinically, infectious abortion was not apparent. Agglu-

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tination tests on the blood serum showed no evidence of abortus agglutinins. Four of the heifers were about seven months pregnant, while the fifth (266) was in her fifth month of pregnancy. (See table I.) Each heifer was observed for a few days after her purchase and temperatures were taken twice each day. As soon as they had become adjusted to their new environment and appeared normal in every respect, each heifer was injected intravenously with 10 cc of a physiological saline suspension of one of the five cultures which had been isolated from the blood of patients showing symptoms of undulant fever. The cultures were grown for 48 hours on serum-agar slants and washed off with a physiological saline solution. With the exception of culture from case J, which was injected into 255, the fourth generation was used. The suspension used for injection of 255 was a mixture of the fourth generation, isolated from patient J's blood when examined the first time, and the second generation of the cultures isolated at the second and third examinations. The density of the suspension injected was measured on the Gates apparatus.[4] The suspension from culture J, which was injected into heifer 255, gave a reading of 1; from culture H, injected into heifer 266, a reading of 1.5; while from cultures W, L and K, it was still more dilute and gave a reading of 2. Small amounts of these same suspensions were injected into guinea pigs to test their pathogenicity. All of the guinea pigs developed typical abortus lesions, the majority becoming emaciated, some of them dying from three to eight weeks later.

The cultures which are designated J, H, W, L and K are from cases showing some variation in the symptoms and course of the disease. The symptoms in the first two (J and H) were typically those of undulant fever except that the course in the former was severe and ran for a period of twelve weeks, while the latter lasted only six weeks and was much more mild. Culture W was recovered from the blood of a patient from which *Bacterium typhosus* was also isolated. The disease syndrome was very atypical of typhoid fever in the beginning but later became more characteristic of the disease. Culture L was obtained from the blood of a man whose condition was diagnosed as subacute bacterial endocarditis. This case had a fatal termination and an autopsy revealed a marked splenomegaly and acute nephritis. The patient from which culture K was isolated had a mild undulating fever for several weeks. His blood showed a four-plus Wasserman and the majority of his lymph-nodes showed enlarge-

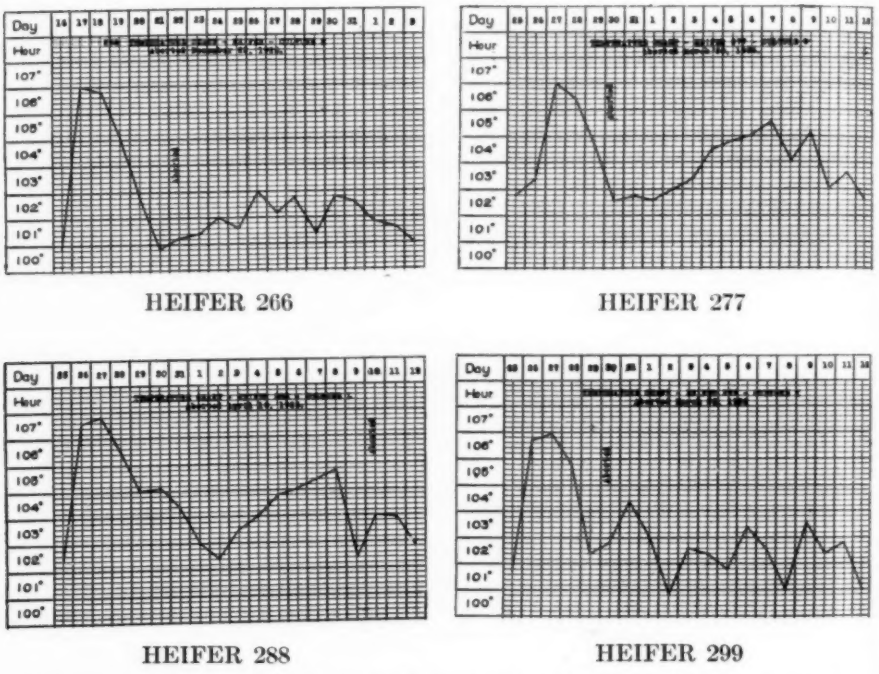
TABLE I—*Results of injecting five pregnant heifers with cultures of Brucella abortus isolated from man*

| Heifer | 255 | 266 | 277 | 288 | 299 |
|---|---|---|---|---|---|
| Days pregnant at time of injection | 204 | 134 | 212 | 220 | 226 |
| Date of injection | 4-10-25 | 12-16-25 | 3-25-26 | 3-25-26 | 3-25-26 |
| Source of culture | Blood Homo J | Blood Homo H | Blood Homo W | Blood Homo L | Blood Homo K |
| Method and amount injected | 10 cc Intrav. | 10 cc Intrav. | 10 cc Intrav. | 10 cc Intrav. | 10 cc Intrav. |
| Date of abortion | 4-30-25 | 12-22-25 | 3-30-26 | 4-10-26 | 3-30-26 |
| Days between injection and abortion | 20 | 6 | 5 | 16 | 5 |
| Date of slaughter or death | 12-17-25 | 2-22-26 | 4-12-26 | 5-10-26 | 5-10-26 |
| Days between injection and slaughter | 251 | 68 | 18 | 46 | 46 |
| Agglutination test on blood at time of slaughter | 1:540 | 1:540 | 1:4,860 | 1:4,860 | 1:14,580 |
| Agglutination test on day of abortion | 1:65,610 | 1:1,215 | 1:60 | 1:43,740 | 1:60 |

ment. The physician in charge of his case had made a diagnosis of syphilis of the lymph-nodes. Some of these tissues were excised and sectioned. The pathologist of Bellevue Hospital, New York City, reported that there was nothing in the histology of the nodes upon which to make a diagnosis of syphilis.

"On the other hand, the changes are such as might easily be in keeping with those produced by *Brucella abortus.*"

The animals were observed frequently each day and temperatures were taken in the morning and evening. Heifers 255 and




HEIFER 266 HEIFER 277

HEIFER 288 HEIFER 299

Temperature charts of four heifers 266, 277, 288, 299, injected intravenously with cultures of *Brucella abortus* isolated from man.

266 were injected at different dates while 277, 288 and 299 received their injections on the same date. It was extremely interesting to observe that a similar reaction was obtained in each animal following the injection of the suspensions. The temperature rose, in a few hours, from normal to 106° and 107° F. and remained at approximately the same level for the first two or three days. (See chart showing temperature curves.)

Respirations became accelerated and the animals showed extreme malaise. They ate only a small amount of food or refused it entirely. As will be seen from the protocols, 255 expelled a

living fetus in twenty days following the injection of the culture, while 266 aborted in six days. Five days after 277 was injected she was observed in the morning and found lying down in her stanchion. She was unable to rise. A vaginal examination showed that the head and anterior limbs of a dead fetus were lying in the vagina. The animal was given some assistance and, by applying traction, the fetus was removed, as well as the placenta. Heifer 288 aborted in sixteen days. The placenta was expelled one hour later. Heifer 299 expelled a living fetus five days after receiving the culture. The placenta was removed for examination very soon after abortion. Inasmuch as we needed calves badly to nurse the cows, the calf was not sacrificed, but it became moribund and was killed and cultured for *Brucella abortus* nine days later.

A bacteriological examination was made of the colostrum and placenta as well as of the lungs, livers, spleens, kidneys, stomach contents and small intestines of the fetuses. Cultures were made on serum-agar and grown in jars in which 15 per cent of the air had been replaced by $CO_2$ as described above. Cultures of *Brucella abortus* were readily recovered from the colostrum and placenta as well as from several organs of each fetus. (See table II.) A small amount of the colostrum, as well as extracts from the tissues mentioned above, was injected into guinea pigs. The lesions produced in these guinea pigs were identical with those produced by the suspensions which were injected into guinea pigs at the time the cows were inoculated. We observed no difference between the atmospheric requirements of the cultures injected and those recovered from the colostrum, placenta and fetus in each case, as well as the cultures from guinea pigs injected with these tissues.

Frequent agglutination tests were made upon the blood serum of the heifers after the cultures were injected. There was a rapid rise of the agglutinin titre of heifers 255, 266 and 288. Numbers 255 and 288 showed their maximum titres of 1: 65,610 and 1:43,740 respectively, about three weeks following their injection. The development of agglutinins in the other two was more gradual and did not reach such a high titre.

All of the animals with the exception of 255 were destroyed in a comparatively short time after aborting. She was allowed to live 231 days. Following abortion each became dull and emaciated and did not respond to the usual methods of treating septic metritis. Heifers 266 and 277 became moribund and were killed.

Numbers 288 and 299 were destroyed 46 days after they were injected. The autopsy on 266 revealed a general lymphadenitis and a pyemia. Cultures of *Brucella abortus* were obtained from practically all tissues examined. (See table II.) Heifer 277 showed at autopsy a general lymphadenitis and some metritis. Heifers 288 and 299 showed no unusual lesions except general

TABLE II—*Results of bacteriological examinations of five heifers and their fetuses*

| Heifer | 255 | 266 | 277 | 288 | 299 |
|---|---|---|---|---|---|
| Colostrum | + | + | + | + | + |
| Placenta | + | + | + | + | + |
| Uterus | — | + | + | + | — |
| Udder | + | + | + | + | + |
| Spleen | + | + | — | — | — |
| Liver | — | + | — | — | — |
| Submaxillary lymph-glands | — | | + | | — |
| Supramammary lymph-glands | + | + | + | + | + |
| Precrural lymph-glands | — | — | — | — | + |
| Prescapular lymph-glands | — | + | + | — | — |
| Sublumbar lymph-glands | | + | + | — | — |
| Mesenteric lymph-glands | — | — | — | — | — |
| Fetus | 255A | 266A | 277A | 288A | 299A |
| Heart-blood | + | + | — | — | — |
| Lung | + | + | — | — | — |
| Liver | + | — | + | — | — |
| Kidney | + | — | — | | |
| Spleen | + | — | — | — | — |
| Stomach contents | + | + | + | + | + |
| Small intestine | + | + | — | — | — |

emaciation. Heifer 255, however, revealed a markedly enlarged spleen, from which *Brucella abortus* was recovered in pure culture.

## DISCUSSION

Basing our opinion upon our present knowledge of the biological characters of *Brucella abortus*, it is evident that these cultures

isolated from human cases of undulant fever do not differ greatly, except in virulence, from the cultures of *Brucella abortus* recovered from other sources. However, certain bovine and porcine strains have shown as marked a virulence for guinea pigs as have those cultures isolated from man. The first generation of the human types has been extremely difficult to grow, but the succeeding generations have shown abundant growth, with the exception of one culture which still requires an increased amount of carbon dioxid. The cultures of human origin are pathogenic for guinea pigs and rabbits. They produce an agglutinin which is absorbed by bovine strains of *Brucella abortus* and they absorb agglutinins from the sera of cattle suffering from infectious abortion. Abortion was produced in pregnant heifers by five of the cultures in a comparatively short time after they were injected. They showed a greater toxicity for the animals than do the bovine cultures when injected intravenously and in each case infection was established in the udder by the human strain injected. After aborting, the heifers did not seem to recover. They remained dull, would not eat well and became emaciated. Heifer 255, which was kept longer than the others, showed some improvement but had not returned to normal approximately eight months later. Large numbers of *Brucella abortus* were still being discharged with her milk. A calf which had been nursing this cow became infected. It was destroyed and autopsied four months after it had been removed from this animal and we were able at that time to recover *Brucella abortus* from its lymph-glands. Its serum also showed complete agglutination when diluted 1:180.

## Summary

1. During the last year the author has studied ten cases of undulant fever in man from which has been recovered an organism indistinguishable from *Brucella abortus*.

2. The diagnosis of these cases has been made by isolating the organism from the blood stream of the patients and by finding the specific agglutinins in their blood serum by the agglutinin-absorption test.

3. An intravenous injection of ten cc of a physiological saline suspension of each of five of these strains was given respectively to five pregnant heifers that did not react to the agglutination test and that were purchased from small herds where infectious abortion was not apparent.

4. The five heifers aborted from five to twenty days after receiving the culture. The organism was recovered from the fetus, placenta and colostrum in each case.

5. One heifer, that was not destroyed until 231 days after aborting, still harbored the organism in her udder and eliminated large numbers in her milk.

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DISCUSSION

DR. W. E. COTTON: I would like to ask Dr. Carpenter whether he tried to inject cattle with the cultures from human sources by feeding.

DR. CARPENTER: No, I have not fed the cultures; I have, by the way, fed milk used by some of these patients, and infected guinea pigs.

DR. COTTON: Some of your heifers aborted so very soon after injection that I am wondering whether the abortions might not have been due, in a large measure, to the severe systemic disturbances that followed the injection of the cultures, rather than to an extremely rapid growth of the abortion organism in the placenta.

DR. CARPENTER: Of course, in those cases where the heifers aborted a short time after the cultures were injected, there is no doubt that the high reaction had something to do with it, but not in all cases. There were lesions of the placenta that were typical of abortus lesions, and the organisms were easily isolated in the cultures. The high reaction probably had something to do with the expulsion of the fetus so early after the injection, and the fact also that they were quite well along in pregnancy, I think, had something to do with it.

DR. COTTON: What proportion of guinea pigs fed infected milk became infected?

DR. CARPENTER: Well, I tried this in milk from only one herd, which was supplying milk to this one patient. I centrifuged the milk and fed the sediment from the milk to either six or eight pigs, and I think there were two or three from that group that were not infected.

I have seen certain bovine strains, although I have not injected them into pregnant cattle, that were very virulent for guinea pigs.

DR. COTTON: Would you say that the human became infected, by chance, with a bovine strain?

DR. CARPENTER: I have no briefs on that, but this I know: The milk that these patients drank contained virulent strains of the organism, that is, more virulent than you usually find in milk supplies; I have studied the milk from several herds and tested the virulence; I have tried it by injecting the culture into guinea pigs and have found extremely virulent cultures in the milk they drank.

DR. COTTON: Do you know whether there is any goat disease in central New York?

DR. CARPENTER: I do not believe there is any goat disease in New York State. I know the Health Department examined the blood from one or two goats and found some positive agglutinins. I do not know whether they were specific for *Brucella melitensis* or for *Brucella abortus*.

DR. COTTON: Were the cultures you isolated from human sources of the bovine or of the porcine type?

DR. CARPENTER: I do not know that I am qualified to differentiate between porcine and bovine strains. I know what Dr. Theobald Smith considers the typical lesions in guinea pigs injected with the porcine strains.

DR. COTTON: You will get joint lesions with both the bovine and porcine strains, but the latter usually cause certain lesions, especially in the liver and spleen and sometimes within the orbit, which the former do not. In

general, they seem to be more virulent for guinea pigs than do the bovine strains.

DR. CARPENTER: I have seen strains isolated from bovines that showed lesions that I could not differentiate from porcine lesions, but perhaps the cattle were infected with a porcine strain. I do not know. I just know they came from bovine sources.

DR. T. P. POLK: Doctor Carpenter, we received some of your strain 80 here at the Experiment Station. I want Professor Good, who is here with us, to tell you about some of the titers he got from some of the blood samples I had brought in at the time when we received the strain 80 from you. While we had these blood samples nearly two weeks before the test was made, still at the same time some of these samples showed a higher titer with your strain 80 than with an antigen made from the Bang strain here. Special pains had been taken to keep the serum from the blood of the cows sterile.

DR. CARPENTER: The strain I sent here was isolated by Meyer, from certified milk. It is not a human strain, but it makes a very good antigen, and that is the antigen we have been using, even on the human case, and so, therefore, I sent along the same strain that we have been using.

DR. POLK: The reason I was so interested was to see if some serum I brought in would agglutinate your human strain. We thought strain 80 was a human strain.

DR. CARPENTER: Did you take the blood serum from these?

DR. POLK: Yes, but I want Professor Good to tell you about this.

PROF. E. S. GOOD: Dr. Carpenter, Dr. Polk thought he had your strain that was isolated from man, so, as a matter of fact, we simply ran it as an antigen in comparison with antigens of our strains and we found the agglutination titer in some cases twice as high with the antigen received from you as we did with the antigen we were using regularly in our tests.

DR. CARPENTER: Did not Dr. Dimock send for that? I thought I told him I was sending him the bovine strain I was using. This strain 80—probably Dr. Cotton is familiar with it—is the best culture that we have ever used for an antigen, and I am very much interested in knowing if you did obtain a higher titer with this strain than you did with any others.

PROF. GOOD: Not altogether. Maybe there were five or six cases in which it went considerably higher than it did with others we were using, but in some cases we found it considerably lower. I could tell by looking up the records. We were interested in it going higher.

DR. COTTON: Dr. Carpenter, the organisms you have obtained from human sources seem to be unusually virulent, as is shown by the short time between infection and abortion and the extremely high titer of agglutinins induced in the blood serum of the infected animals. What is the highest titer you obtained?

DR. CARPENTER: One, to sixty-five thousand and another up to forty-three thousand.

DR. COTTON: Have you ever obtained as high a titer with a strictly bovine strain?

DR. CARPENTER: Well, I have not followed that closely. I can not say whether they went as high as that or not.

DR. COTTON: In our work I think the highest titer we have obtained with blood is about one to sixty-four hundred. We have had colostrum go as high as one to twenty-five thousand, but I do not remember any blood sample exceeding one to sixty-four hundred. In infections with your strains from human sources it goes so very much higher, and you get your abortions so very quickly, five or six days after an injection. There seems to be a very wide difference between these strains, and the bovine strains with which we are familiar. Of course, I know bovine strains do vary, but I have not known them to do so to this extent.

DR. J. G. HARDENBERGH: Did these cases of infection of students occur on farms, from drinking unpasteurized milk?

DR. CARPENTER: Three of these cases have occurred in students after entering Cornell University, and another occurred in a fifteen-year-old boy in Ithaca. None of these men have been on farms at all. Another case is a minister. That occurred over in Kingston, N. Y., and he has not been on a

farm at all. The others were cases, as I told you, from Bellevue Hospital, in New York City. One patient's case was a laboratory infection, undoubtedly because he had been working in a laboratory with *Brucella abortus* of bovine origin, and had not been working with swine or goats. None of them had been on farms whatsoever. There was one, the second case, H, who spent his summer on the farm, but he was a graduate student in the University at the time of his illness. He came back in the fall after spending a summer on the farm. He came back in September, and his symptoms did not begin to show up until November. Whether he had picked up this infection at that time I do not know, but he had nothing to do with swine whatsoever, or had not seen any swine. We have done considerable work, trying to find out if these men had anything to do with swine, and so far as we can learn, not a single case has been associated with these animals.

DR. I. D. WILSON: What about the history of the infection?

DR. CARPENTER: We have studied it very closely in two herds where they had much abortion. They do not seem to be able to clean it up. The animals abort repeatedly, and the infection seems to be extremely virulent.

DR. COTTON: Have you compared the organisms infecting these herds with those from human sources?

DR. CARPENTER: Yes, and we find them quite similar but, of course, the lesions vary with the amount of milk you inject, or the amount of culture you inject. I did not answer your question as directly as I should, however, but I know that Dr. Smith, who has worked with five of these strains, is of the opinion that they are swine strains, and they produce large lesions in the liver of the guinea pig, and large spleen. You also get enlarged lymph-nodes.

DR. COTTON: At the Experiment Station, we injected a number of guinea pigs with a strain of *Brucella melitensis* supplied by Miss Alice Evans, of the Public Health Service. It was one of the strains that she used in her work in causing abortion in cattle with the Malta fever organism. I believe that she called it the Phoenix strain. The lesions caused by it in the guinea pigs were very similar, if not identical, in appearance with those caused by *B. abortus*. They were not of the porcine type, but there was a suggestion of it in the form of certain very small, spherical, white nodules in the liver.

DR. CARPENTER: Is that the one she said came from Arizona?

DR. COTTON: Yes.

DR. CARPENTER: How early do the lesions occur in the guinea pig?

Dr. Cotton: Of the guinea pigs infected with the melitensis organism, a small number died a month or less after injection and one was killed at the end of a month. The remainder were allowed to live a little more than two months and an autopsy showed well-marked lesions. The guinea pig killed at the end of a month showed early lesions similar to those of abortion, but those dying of intercurrent disease did not. We usually get fairly well-marked lesions with *B. abortus* in six weeks and sometimes at the end of a month, but make a practice of allowing the animals to live two months.

DR. B. T. SIMMS: Have you found that there is a distinct relation between the capacity or virulence of the organism in the various types of animals? That is if you have an organism that is particularly virulent for cattle, will that be particularly virulent for guinea pigs? That is, where you are trying to produce abortions in cattle. Perhaps we do not have a virulent strain. If you infect a guinea pig, is it necessarily virulent for cattle?

DR. CARPENTER: That is the experience we have had in cattle. If we isolate a strain from a herd that is having many abortions, it is more liable to produce marked lesions in guinea pigs than a strain isolated from herds having sporadic cases.

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The wills, the won'ts, the can'ts—just three classes of men. The first accomplish everything, the second oppose everything, the third fail in everything. Classify yourself.

# GENITAL INFECTION IN MARES BY AN ORGANISM OF THE ENCAPSULATUS GROUP*

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In our work on the pathology and bacteriology of the repreductive organs of mares and their relation to sterility, special attention has been given to the study of microorganisms isolated from mares suffering with cervicitis and metritis.

Among the microorganisms isolated is one belonging to the encapsulatus group. The recovery and identification of this organism from cases of metritis was reported in the Annual Report of the Kentucky Agricultural Experiment Station, Part 1, 1924, and the organism was designated *Encapsulatus genitalium.*

Morphologically and culturally this organism is apparently indistinguishable from *Encapsulatus inguinalis,* Friedländer's bacillus and others of the encapsulatus group; however, there seems to be some difference biochemically and in habitat and, according to the results of our experimental work with this and other organisms of the encapsulatus group, they are quite consistently different in pathogenicity.

Cultures have been made from the cervix and uterus of 1424 mares. Out of this number, the microorganism which we have designated *Encapsulatus genitalium* has been recovered from 62 mares, or 4.4 per cent of the total number cultured. During the last twelve months we have had cultures from 806 mares. Of these 511 were mares not previously examined, while 295 were mares that had been cultured previous to 1925. All the mares from which this organism was recovered showed clinical evidence of cervicitis and metritis with marked pathological exudation. All the mares except three had been barren at least one year, and were found to be infected after the close of the breeding season, which extends from February to June.

The organism was first recognized as a cause of metritis in October, 1923. If recovered previous to this date, it was considered as a secondary invader or as an accidental contamination of the culture tubes; however, none of the mares cultured previous to the fall of 1923 and of which we have subsequent records, except those that had been exposed to the infection, later showed

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infection with this bacillus. Therefore, we feel that the organism was not overlooked in our cultural work on cases of metritis previous to its isolation and recognition from case 2134, October, 1923. Of the 60 cases that have come under our observation the history of at least 50 per cent is known in detail. In all the cases of which we have a definite history, infection occurred as a result either of direct contact from the stallion, artificial insemination, or from treating mares for sterility along with those infected with *Encapsulatus genitalium;* hence, by the hands of the operator, instruments and solutions used.

## Clinical Pathology

The presence of *Encapsulatus genitalium* in the genital tracts of mares results in the development of inflammatory processes that are clearly evident upon clinical examination. Externally, the hairs of the tail usually are soiled and matted, and dried exudate will be observed on the quarters adjacent to the external genitals. Upon rectal examination the walls of the uterus feel abnormally thick and the organ as a whole is enlarged. In these cases where there is an excessive accumulation of exudate, the uterus is distended in proportion to the amount of exudate present. In the case of old mares that have had several foals, and in which the uterus has become more or less dilated, pendulous and atonic, or in cases where adhesions have formed in the cervix, the exudate accumulates in the uterus in great quantities.

In a few cases the distention has been such that the condition might have been mistaken for early pregnancy; the uterus is, however, decidedly atonic, soft and doughy as compared with a physiological, gravid uterus. In younger mares in which the uterus is well contracted and has retained its normal tone, the exudate which forms from the action of the *Encapsulatus genitalium* apparently is discharged from the genital tract as fast as it forms. In general, it has been our experience to find that the conformation of the cervix and uterus is not so apt to be distorted as in the case of cervicitis and metritis due to other types of infection.

The differential diagnosis of cervicitis and metritis in mares due to *Encapsulatus genitalium*, *Streptococcus genitalium* and other microorganisms should always be confirmed by a bacteriological examination. There are, however, certain changes rather typical and characteristic of this infection. First and most important of all is the character of the exudate. The exudate

always is thick, often containing many flocculi, exceedingly viscid and tenacious in consistency and slimy to the touch. The color varies from a dull gray to a yellowish white. The visible portion of the cervical membrane has a dull reddish-brown color.

During the period of estrus, exudation is profuse in all cases. In some cases, during the interval between the periods of estruation, the condition of the genital tract may return to a state closely resembling normal. However, by manual examination of the genital tract the slimy exudate will be recognized and cultures are invariably positive regardless of the time when this may be taken.

### Gross Pathology

The pathological changes are those of congestion, inflammation, exudation, degeneration, desquamation and proliferation.

The gross pathology of cases as they are met with in the field varies somewhat in the extent and character of the lesions. This variation is due in part to the condition of the genital tract of the mare at the time she becomes infected. In a number of cases studied the mares were suffering with metritis due to other causes before they became infected with *Encapsulatus genitalium*. In practically all such cases the lesions are found exaggerated over those observed in mares that were apparently normal when infection took place. Other conditions which tend to modify and intensify the lesions are age, length of time infection has been present, time of estrual cycle, number and frequency of services during the breeding season and, finally, the treatment that may have been employed. The inflammatory changes usually involve the entire genital tract; however, the main seat of pathological change is seen in the cervix and uterus. The vaginal mucosa apparently is very resistant to this and other forms of bacteria. The secretion of exudate from the cervix and uterus, teeming with infection as it passes out over the vaginal mucous membrane, is naturally a source of great irritation but does not result in any serious inflammatory processes. Upon control of the uterine secretion the vaginal membrane returns to normal.

The cervix always is involved in the inflammatory processes and contributes freely to the formation of exudate; however, the extent to which the cervix becomes distorted varies greatly in different cases, as stated under clinical pathology. The characteristic color of the vaginal cervix, as seen upon clinical examination, is a dark brownish-red; upon opening the cervical canal on post-

mortem examination the color is found to be of a dark purplish-red. The cervical folds are thickened from inflammatory processes and covered with the characteristic slimy exudate. While the epithelial lining of the folds and of the excretory ducts and glands always is more or less involved, it is quite apparent that the muscular and fibrous structures do not always become seriously thickened through inflammatory processes. In some cases the whole cervical structure has been found to be greatly enlarged. To know in just what proportion the enlargement is the result of infection with *Encapsulatus genitalium* or to causes operative previous to infection would require that the condition of the genital tract of the mare be known previously to the time infection took place.

## METRITIS

Upon opening the uterus the viscid exudate and dark purplish-red areas of discoloration stand out in great clearness as evidence of disease. The amount of exudate varies, depending upon the severity of the infection, but apparently more directly upon the condition of the uterus at the time infection took place. In what might be considered the average case, from 200 to 400 cc of the exudate will be found. The exudate is of a yellowish-white or dull-gray color, often very frothy and containing numerous flocculi and many cells of inflammatory and degenerative origin.

In the beginning, the process apparently is a typical endometritis. The whole surface of the uterine cavity is involved; the changes however are always more severe in the body of the uterus than at the apices of the horns. The infection apparently remains more or less superficial, for while the walls of the uterus are thickened through inflammatory processes, culture media inoculated from the deeper layers after removing the mucous membrane remain sterile.

Often there are irregularly-shaped hemorrhagic areas measuring from one to three centimeters in diameter, the outline being more or less easily traced. In cases of long standing the structures below the mucosa become involved through chronic inflammatory processes, resulting in fibrosis with a corresponding loss in and arrangement of the structures and tissues normally making up the various layers of the uterine wall.

In some uteri examined, numerous submucous cysts are found. Cysts of a similar character have been observed in other conditions and are therefore probably not a part of the pathology

resulting from infection with encapsulated bacilli. Culture tubes inoculated from the cyst contents fail to show bacterial growth even in those cases in which microorganisms are present in the uterine exudate in great numbers.

### Salpingitis

It has been our experience to find that inflammation of the oviducts is rare in mares. In a few cases a small amount of thick, yellow exudate has been found, rarely in quantity sufficient to occlude the tube completely. The successful passage of sperm cells would be doubtful, even in cases of mild inflammatory changes. In no case have the fallopian tubes been found enlarged, even in those cases where the infection had involved the tubes and ovaries. No gross lesions were found in the ovaries that could be attributed directly to this infection. In one case there was considerable inflammation and adhesions between the fimbriated end of the fallopian tube and the ovary.

Pathological changes found upon microscopic examination of sections of the uterus in the main substantiate the gross pathology. The sections examined were taken from several parts of each uterus. In each case the sections studied showed about the same degree of abnormality. This indicates that the inflammatory process is quite uniformly distributed throughout the uterus. Of all the different structures of the uterus the mucous membrane presents the most noticeable changes. Areas will be found in which the mucosa remains intact and shows only slight degeneration of the cells. The cells have a cloudy, granular appearance. The general outline of the cell may be lost and cannot be separated from its neighbor. Nuclei of the epithelial cells in these areas are seen in the lower part near the basement membrane. They also lose their distinctness of outline. In those areas where the epithelium is still intact, the submucosa, glandular and muscular layers are not severely involved.

In other cases degeneration has progressed to such an extent that the mucosa is destroyed completely and sloughed. The exudate associated with these areas contains mucus, degenerated epithelium, cell detritus, pus cells and inflammatory round-cells in the reticular stroma immediately underlying the epitheliun, especially in regions where there is a break in the continuity of the mucosa.

The deeper muscular layers which appear upon sections as twisted, irregularly-shaped bundles running in all directions may

be still well-preserved but that they are later to become seriously involved in the inflammatory process is indicated by the presence of products of inflammation found scattered throughout all layers of the uterine wall.

The blood-vessels principally affected are those in the muscularis mucosa. They are greatly distended with blood and around each vessel is a zone of inflammatory round-cell infiltration. Another rather frequent condition existing is an edema in the submucous area.

In sections from cases of a more chronic nature the pathological picture is that of a typical chronic productive inflammation. Many areas will be found where the normal columnar epithelium of the mucosa has been replaced by a stratified squamous type.

### Uterine Alterations

The stratum nominally recognized as the submucosa and muscularis mucosa and in which the uterine glands are found has become very largely obliterated during the course of the inflammation and is seen as a mass of newly-formed connective tissue. In the deeper muscular layers of the uterine wall the inflammatory process has been equally active. In some sections a typical granulation tissue is found. In others the inflammatory tissue is of a still more advanced type and has approached a state bordering on fibrosis. The newly-formed connective tissue penetrates the entire uterine wall from mucosa to serosa and has more or less completely displaced and obliterated the different layers and bundles of muscle tissue as normally observed in sections of the wall of the uterus.

Microscopic sections of the cervix show changes similar to those noticed in the uterus, except that they are, as a rule, more severe.

The surface epithelium and that lining the secretory glands undergo most severe inflammatory and degenerative changes, often resulting in complete destruction. The lumen of the glands may contain a thin mucus-like substance intermixed with cells and detritus or may be packed and distended with a solid homogeneous mass. The opening of the gland ducts may be closed as a result of regeneration of the surface epithelium. The stroma often is involved in the early stages, showing changes of an acute inflammatory nature, later becoming enlarged and thickened in the form of a true hyperplasia.

The pathological changes of the fallopian tubes are limited mostly to a catarrhal salpingitis. In different sections of the

same tube and sections of different tubes the changes noticed are quite uniform and mild in character. The lining epithelium shows a degeneration of a mild type. The cell bodies break down into a granular mass with poorly-staining muclei. A mild infiltration by round cells sometimes is seen in the submucosa. The remaining muscular layers and serosa appear normal. An exudate of mucus containing round cells and degenerated epithelium is often present in the lumen of the tube. Large, irregularly-shaped, homogeneous masses of uniform staining qualities are seen here quite often. This material is similar to that noticed in the cervical glands.

The etiological factor in this condition is a non-motile, encapsulated rod. The morphology of the organism is variable. It varies in length from 1.8 to 3.7 microns and in breadth from 0.9 to 1.7 microns. Long rods and coccoid forms may be encountered in the same smear. Especially is this variability of size and form noticeable in young cultures. In older cultures the organisms tend to assume a coccoid form.

### Cultural Characteristics

The bacterium stains readily with the ordinary anilin dyes, is Gram-negative and does not produce spores. The capsules are most easily demonstrable in animal exudates, particularly the peritoneal fluid of guinea pigs killed by intraperitoneal injection.

The outstanding characteristic of the organism is its abundant slime-production on all media.

On agar slants there occurs an excessive dirty-white to slightly-yellow growth, which is moist, spreading, glistening and very viscid. Agar colonies are large, raised, round and entire. Broth becomes turbid with a ropy sediment. A typical nail-head growth appears in gelatin. No liquefaction occurs. Nitrates are reduced to nitrites and ammonia. Indol is not formed.

The above characters place the organism in the genus Encapsulatus. Several species have been described which belong to this genus. Most prominent among these are *Encapsulatus pneumoniae* (Friedländer's bacillus), *Encapsulatus inguinalis* (bacillus of inguinal granuloma), and *Encapsulatus rhinoscleromatis.*

These organisms have been studied by a number of workers, but as yet no satisfactory method of differentiation has been found. The differences between individual strains of the same species are so great that it has been found impossible to separate the group on the basis of laboratory tests. In order to determine

the identity of the organisms which we have isolated from mares a collection of strains of the mucosus group has been made and a comparative study is being carried on. This collection is composed of 25 strains of equine origin, 28 strains of Friedländer's bacillus, 18 strains of bacillus of inguinal granuloma, 2 cultures of *B. rhinoscleromatis*, and 5 cultures of *B. aerogenes* which possess a mucoid form of growth.

While the bacteriological studies are by no means complete, it seems desirable to give a progress report of the work at this time.

Up to the present the bacilli have been subjected to the following comparative tests: Their nitrogen and carbohydrate metabolism has been studied; they have been subjected to agglutination by antisera prepared from various members of the group; precipitation tests have been carried out; their pathogenicity has been studied, and their ability to produce cervicitis and metritis in mares has been tested.

## Cultures Unusually Uniform

The fact which has been most emphasized during the course of the investigation is the unusual uniformity of the cultures recovered from different mares. The twenty-five equine cultures being studied have been recovered from a number of studs in Kentucky and Virginia. As far as we have been able to determine, all these cultures are exactly identical. This is surprising, since other investigators have found a decided lack of uniformity in encapsulated bacilli recovered from a single source. This variability in Friedländer and granuloma strains has been very apparent in this work.

None of the organisms of this group produces indol. When tested for their ability to grow in Koser's citrate medium, all the equine strains were found to be able to utilize citrate as a source of carbon. Twenty-two of 27 Friedländer strains were able to initiate growth, while citrate fostered the growth of only 7 of the granuloma cultures. All the equine strains were methyl-red-negative and Voges-Proskauer-positive. Nine of the Friedländer strains were positive and 18 negative to methyl red. Sixteen gave positive Voges-Proskauer tests and 11 were negative. Of the granuloma cultures 3 were methyl-red-positive and 15 were negative. Among the Friedländer bacilli there was a fairly close correlation between the methyl-red and Voges-Proskauer tests. No such correlation was observable among the granuloma strains.

When the action of the organisms on the carbohydrates is considered, the constancy of the equine strains is again apparent. All the organisms ferment dextrose, lactose, sucrose, raffinose, xylose, salicin, glycerol and adonitol, with the production of acid and gas. Dulcitol and inulin are not attacked.

In the Friedländer and granuloma strains the fermentation reactions are very markedly variable. Almost every possible combination of fermentative reactions has been met with. Some of the strains are very active fermenters, while others are apparently non-saccharolytic.

The serological work has not as yet progressed far enough to warrant drawing any definite conclusions as to the antigenic identity of the equine cultures. All the equine strains apparently belong to the same agglutinative type; that is, they are all agglutinated by the same antisera. This is also borne out by precipitin reactions. The Friedländer and granuloma strains are heterogeneous in their agglutinative character, the same variability being exhibited in this way as in others.

### Cross-Agglutination Tests

Certain of the Friedländer strains cross-agglutinate with the equine cultures in rather high dilution. Some of the cultures of aerogenes being studied also cross-agglutinate with the equine cultures and with certain of the Friedländer strains. It is impossible to determine by simple agglutination how closely these cultures are related. It is hoped that this question may be determined by the application of agglutinin absorption.

The pathogenicity of the equine strains for the laboratory animals is much the same as that of Friedländer's bacillus and the bacillus of inguinal granuloma. Guinea pigs and rabbits, when injected intraperitoneally, develop septicemia and die in 12 to 24 hours. Rabbits injected intravenously with relatively large doses of heat-killed suspensions often die within 24 hours.

In mares the pathogenicity of the equine cultures is very different from that of any of the other strains which have been tested. It is possible to reproduce the disease regularly by intra-uterine inoculation of saline suspensions of organisms recovered from mares. Within a few days after inoculation the mares develop cervicitis and metritis, with a copious, slimy exudate, containing flocculi, leucocytes and tissue detritus. The organisms may be recovered in great numbers by culturing the os uteri.

During the past year postmortem examinations were held on four mares infected with encapsulated bacilli. In all of these cases the organism was recovered from the cervix, body of the uterus and the left and right horns. In one of the mares (4131) negative cultures were obtained from the fallopian tubes and ovaries. In the second case (2444), a mare that had become infected naturally, the organism was recovered from the left tube and left ovary. In the two remaining mares (4132 and 4233) the bacilli had invaded both the left and right tubes and the left and right ovaries. In one of these latter cases (4132) the bacterium was recovered also from an inguinal lymph-gland.

When suspensions of Friedländer's and granuloma bacilli are placed in the uterus the results are quite different. Even if the whole of an agar-slant culture be introduced through the os uteri, the mares do not become infected. Negative cultures can be obtained from the cervix after three to five days. These mares remain negative on continued cultures and show no infection of the genital tract on autopsy.

The infection is transmitted readily through breeding. An infected mare (4141) was bred to a stallion known not to be a carrier of the organism. Following this he was allowed to serve two mares (4133 and 4132). Both these mares had given repeated negative cultures. Within a week both of them had developed cervicitis and metritis, with a slimy exudate. Numerous colonies of the organism appeared upon the cultures taken from the cervix.

The attached case reports give evidence of the transmission of the infection through breeding under field conditions.

*Case No. 3090:* First examination, November, 1924. Cervical folds thickened and protruding into vaginal cavity, characteristic slimy exudate plainly evident upon clinical examination. Cultures from cervix and uterus positive to *Encapsulatus genitalium*. Second examination, February 1925. Cervix about normal in size, no exudate in vaginal cavity although membrane slimy to touch. Unable to pass platinum needle through cervical canal. Manual examination showed cervix closed by adhesions. Rectal examination revealed that uterus was distended with exudate. Upon postmortem examination two gallons of an extremely viscid and most tenacious exudate was found in the uterus. Culture tubes inoculated from uterus positive to *Encapsulatus genitalium*.

*Case No. 2390:* First examined, January 1924. Cervix not enlarged. Cultures from cervix and uterus positive to streptococcus. Treated for metritis from January to May, 1924. Bred once by artificial insemination, spring of 1924. Examined for pregnancy, September 1924. Barren. Culture tubes inoculated from cervix and uterus positive to streptococcus. Treated from September 1924 to February 1925. Bred several times during spring of 1925. Examined for pregnancy, fall of 1925. Barren. Treated during winter of 1925 and 1926. Culture tubes inoculated from cervix and uterus, April 1926, positive to *Encapsulatus genitalium*. Treatment continued. Cultured, May 1926. Positive to *Encapsulatus genitalium*. At time of last examination cervix

small, hard, rigid and tube-like and lined with a smooth, shining membrane continuous in its make-up, with no evidence of mucous folds. During all this time the cervix had remained about the same in substance of connective and muscular tissue. The principal change was apparently in the mucous membrane and glandular structures.

*Case No. 2282:* Bred spring of 1923. Nov. 20, 1923, examined for pregnancy. Barren. Dec. 7, 1923, clinical examination of genital tract showed mucous membrane congested; os relaxed, flaccid, foamy exudate in vagina. Culture tubes inoculated from cervix and uterus—yielded a streptococcus. March 7, 1924, culture tubes inoculated from cervix and uterus—yielded a streptococcus. Kept under observation and treatment until May 6, 1924. Improved. Bred late in May. Fall of 1924, examined for pregnancy. Barren. Oct. 23, 1924, purulent exudate in genital tract, vagina, cervix and uterus. Culture tubes inoculated from cervix and uterus, positive to *Encapsulatus genitalium*. Oct. 26, 1924, cultured. Positive to *Encapsulatus genitalium*. Last treatment and examination, Nov. 6, 1924. Spring of 1925, bred. Conceived and gave birth to an apparently normal foal, May 1926. Period of gestation 12 months. Bred on ninth day, came back in season in 18 days, bred and is at present apparently in foal.

*Mare No. 2282:* Apparently became infected with *Encapsulatus genitalium* during the breeding season of 1924. This is the only case of a mare infected with *Encapsulatus genitalium*, of which we have a record, that has produced a foal. A number of other cases that have been under treatment have been bred this last spring but to date there has been no opportunity to determine how many of them, if any, are in foal.

*Cases 2134 and 1459:* Mares 1459 and 2134 were both bred to the same stallion in the spring of 1923. Mare 2134 had not been previously examined. Mare 1459 was not previously infected with *Encapsulatus genitalium*. It is assumed that these mares were infected during the breeding season.

Three other mares (2359, 2268 and 2270) were negative to *Encapsulatus genitalium* on previous examination. They were under treatment for metritis together with mares 1459 and 2134 during the fall of and winter of 1923-24. Mare 2359 first showed viscid rod, Feb. 25, 1924. Mare 2270 first showed viscid rod, March 14, 1924. Mare 2268 first showed viscid rod, April 28, 1924. It is assumed that the infection was transmitted to these three latter mares through the process of treating, either by the hands of the operator, the instruments used, or possibly through the solutions used in washing the external genitalia.

*Case No. 2333:* Age, 3 years. Bred for first time in spring of 1923 by artificial insemination. Conceived. Dec. 20, 1923, aborted. Dec. 24, 1923, examined and cultured (cervix and uterus). Culture tubes positive to *Streptococcus genitalium*. March 23, 1924, cultured. Positive to *Ps. pyocyanea*. May 26, 1924, cultured. Rod not identified. Bred late in spring of 1924. October 1924, examined for pregnancy. Barren. Culture tubes inoculated from cervix and uterus positive to *Encapsulatus genitalium* and *Ps. pyocyanea*. Mare not treated so far as known. Bred, May 5, 1925. Nov. 30, 1925, examined for pregnancy. Barren. Culture tubes inoculated from cervix and uterus positive to *Encapsulatus genitalium*. Treated during winter of 1925-26. May 21, 1926, apparently normal clinically. Culture tubes negative. Bred, May 21, 1926.

May 5, 1925, bred to stallion 63. Mare 3848, foal at side, bred to stallion 63, May 6. 1295. Mare 3849, foal at side, bred to stallion 63, May 7, 1925. Mares 3848 and 3849 examined, Nov. 30, 1925. Both barren. Clinical evidence of metritis. Culture tubes inoculated from cervix and uterus positive to *Encapsulatus genitalium*. Both mares treated for metritis.

Mare 3848, cultured Jan. 1, 1926. Negative. Cultured Jan. 21, 1926. Negative. Cultured Feb. 20, 1926. Negative. Cultured April 6, 1926. Streptococcus. No further record. Not bred in spring of 1926.

Mare 3849, cultured Jan. 21, 1926. Positive to *Encapsulatus genitalium*.

Mare 2359 is known to have been infected with *Encapsulatus genitalium* since February 1924. She was bred to stallion 72 early in the spring of 1925.

The mare failed to conceive at the first service and was bred to the same stallion a number of times during the season.

Mares 2390, 2444, 2445 and 2927 were also bred to stallion 72 during this season. Since the mares failed to conceive they were bred repeatedly. When examined in the fall of 1925, they were all found to be infected with *Encapsulatus genitalium*.

Mares 5241, 5242, 5243 and 5244 were cultured for the first time, Aug. 3, 1921. All of these were infected with *Encapsulatus genitalium*. This latter group of mares had been bred to or impregnated from stallion 72 during the breeding season of 1925 and 1926.

The field cases cited here give evidence of the severity of the infection and the ease with which it may be transmitted through breeding. These facts are confirmed by the results of the breeding experiments which have been referred to above.

ACKNOWLEDGMENTS

The writers wish to acknowledge the work of Dr. E. A. Caslick, who took an active part in the investigation during the three years he was connected with the Experiment Station. Mrs. Ethel Snyder Steiner was assistant bacteriologist in the Veterinary Department of the Kentucky Experiment Station at the time the organism was first isolated. Also our thanks are due to Dr. J. F. Bullard, assistant veterinarian in the Department, who has helped materially in the pathological work.

## MISSOURI SHORT COURSE

The Missouri Veterinary Medical Association, in conjunction with the Veterinary Department of the University of Missouri, will give a four-day special course for graduate veterinarians, January 25-28, inclusive, at Columbia, Mo. This project was tried out about a year ago for the first time in Missouri and sixty-five veterinarians took advantage of the opportunity. Those charged with making up the program for the course this year hope to make it even more attractive than last year. Further information may be obtained from Dr. J. D. Ray, secretary of the Missouri Veterinary Medical Association, 400 New Centre Bldg., Kansas City, Mo., or Dr. J. W. Connaway, University of Missouri, Columbia, Mo.

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To find delight in one's business, to begin it anew each day, to build always for the future, to glorify one's work and be glorified by it—these things lead to the pleasant places among the sunlit fields men call the Garden of Success.—*Will Judy in "Fifty Essays About Men and Things."*

# SOME OBSERVATIONS ON THE OESTROUS CYCLE AND REPRODUCTIVE PHENOMENA OF THE MARE

## A Preliminary Report—Second Paper*

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The entire field included in the above title is so broad that one could not attempt to cover it in the time available nor do we claim to be able yet to discuss all the phenomena of the oestrous cycle or of reproduction in the mare. We therefore will limit this discussion to our observations on the occurrence and duration of oestrus and especially to changes in the ovaries of the mare during the dioestrous cycle and during pregnancy.

When one reviews the literature it is surprising how little research has been done and how little is known about the oestrual phenomena of the mare. Much of our supposed knowledge has been borrowed from better-known species, such as the cow, sow and smaller animals, and unfortunately it is misleading in some respects when applied to the mare.

Among those who have contributed to the literature are Heape[1] who, in 1900, gave some general observations indicating (a) that the mare is a "polyoestrous animal with a tendency toward monoestrum," (b) that the breeding season may last from one to eight months, and (c) that the dioestrous cycle lasts three to four weeks.

Marshall,[2] in 1922, stated (a) that "the normal dioestrous cycle is about three weeks," (b) that "the oestrous period is one week, though its actual length may vary by three or four days" and (c) that "the sexual season extends throughout the spring and early summer."

Ewart,[3] in 1915, believed the period of oestrus to be shorter later in the season and to disappear when the food became scarce or less nutritious. He states that "under favorable conditions, however, mares may become pregnant in the winter."

Seaborn and Champy[4] reported, in 1923, some of the first actual research on the mare's oestrous cycle. They concluded that "rut lasts 6 days at intervals of 3 to 4 weeks" and that "heat shows two periods, a preliminary one of refusal and delay and a subsequent one of acceptance, three days each." Later,

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Seaborn[5] stated that the cycle lasts 24 days, including (a) three days of prooestrum, in which the signs of heat were well marked but the mare was unwilling to accept the male, then (b) three days of oestrus. The balance of the cycle is given as (c) about ten days of metoestrum and (d) about eight days of rest. This report is based on findings made in France, on mares tried with a stallion and destroyed at different stages of the cycle, so that the reproductive organs might be studied, a method which, because of the marked individual variations, would be very confusing.

Our method of study has avoided these difficulties by following the cycle in normal living mares, (1) by rectal palpation of the ovaries and uterus, usually at intervals of one to three days, depending upon the stage of the cycle; (2) by regular teasing with a stallion; (3) by direct examination of the vulva and (4) by the occasional use of a vaginal speculum. The greatest difficulty with this system is that it does not permit measuring or weighing the organs, which makes it necessary that these be given as estimates.

Many mares have been examined once or oftener. Thirteen mares have been palpated regularly for the duration of a cycle or longer and five pure-bred draft mares have been constantly under our observation, from eight to thirteen months. That they are normal is shown by the fact that each has been pregnant part of that time although one (XIV) was held open for nine months. We have also secured and studied many ovaries and genital tracts from oophorectomies and autopsies but many of these have no definite history.

We have found the interval between the onset of heat periods to vary from 20 to 25 days, although in two cases it was about 35 days. The average is 22 or 23 days. The duration of heat is also very variable and many continue anywhere from 4 to 11 days,* averaging about 7 days. There is some variation in periods of the same mare and a decided variation between periods of different mares. Most mares are willing to accept the stallion at any time during heat and fail to show any external signs which could be interpreted as a definite prooestrum although some mares (XXV) will for several days, both before and after heat, throw off urine while actively hostile when teased by a stallion.

Our observations have indicated that the mare has no limited breeding season and that the length of the heat periods is little

*Mare XXIII, in her first oestrus after abortion, was in heat at least 20 days.

if at all affected by weather conditions. When kept in an unheated stable, on scarcely a maintenance ration of oats and poor hay, mare XIV came in heat regularly throughout the fall and winter, her heat periods being constantly about seven days. Mare XXVI, who was in heat when secured on December 1, 1925, returned to heat from December 20 to 30, an eleven-day period during an extremely cold spell. She accepted service on the second, fifth, tenth and eleventh days of heat and conceived. However, before it can be definitely stated that mares do come in heat regularly and do have normal periods throughout the winter, observations should be made on a larger number with little or no shelter.

We believe that the only reliable method of detecting heat is by using a stallion or a teaser. Smears taken from the wall of the vestibule have failed to show any dependable cyclic changes. Changes occurring in the vulva are sometimes quite definite but are not constant. The classical picture of heat is a vulva which is relaxed, with the mucous membrane reddened and moistened with mucus. This mucus is usually noticed as strings or bubbles and is seldom present in quantities sufficient to be thrown off. However, this condition is inconstant even during heat. The uterus usually feels thicker-walled and seems to have more tone during heat but again this change is not constant and sometimes not very marked. The ovaries, by their development of follicles and corpora lutea, give the most reliable information as to cyclic changes but even they do not indicate the onset or termination of heat.

When we go to the literature for information on the mare's ovaries we find little except anatomical descriptions. However, Seaborn and Champy [4,5] tell us that the size of the ovaries varies greatly with the oestrous cycle, being much increased during heat. This they found was caused mostly by the development of follicles but partly by a gorging of the lymphatics with a liquid like that in the follicles. The latter was present during heat even in the occasional absence of well-developed follicles. They concluded that a follicle is mature but never broken on the fourth day of heat and ruptures on the fifth or sixth day. They found that mature follicles contain from 50 to 80 grams of liquor folliculi.

Our method of combining the use of a stallion with rectal palpation has enabled us to determine accurately just when ovulation, the climax of every dioestrous cycle, occurs. Further-

more by palpating ovaries just before oophorectomy or autopsy and by measuring and examining these same ovaries directly after taken, one can learn to recognize structure and become fairly exact in estimating dimensions. To simplify indicating the size of the ovaries we have carefully measured and weighed several, so that when given their dimensions, we can estimate their weight and thus give their relative size in one figure. The sizes given with the following observations are, therefore, not exact but we believe they are reasonably accurate.

With heat lasting from 4 to 11 days there is a possibility of wide variation in the time of ovulation but we have found it occurring usually during the last or next to last day of heat. Ovulation seemed to fall with about equal frequency on the last two days in periods of 7 days or less and to fall on the next to last day, or occasionally a day earlier, in periods of more than 7 days. In other words ovulation has been noted from the 4th to the 10th day of heat.* Breeding seems to have no effect on ovulation nor on the duration of heat. It is evident that the length of the cycle between two ovulations may vary from the cycle between the onset of the two corresponding heat periods but the average of several of these synchronous cycles will be the same. The period between successive ovulations has varied from 19 to 28 days but averaged 22 or 23 days.

Ovulation does not necessarily alternate between the ovaries. A chart of 19 successive cycles including 28 ovulations—20 single and 4 double—from 5 mares shows ovulation alternating 9 times, repeating in the same ovary 6 times, and doubling four times. Three of the double ovulations were one from each ovary, on successive days, the other had both from the left ovary on the same day.

The size of the ovaries varies a great deal, both with different stages of the cycle and in different mares. When two mares of equal size are compared at similar stages throughout the cycle it may be found that the ovaries of one may average as much as 50 per cent greater in dimensions† than the ovaries of the other. Then the same ovary may change enough in one cycle to make its volume from two to seven times as large just before ovulation as it was earlier in the cycle. The great size attained just before ovulation is due (a) chiefly to the mature follicle, or

*Mare XXIII, previously referred to, ovulated on the nineteenth day, was then bred and conceived.

†A 50 per cent increase in all dimensions is equivalent to an increase of nearly 3½ times in volume; likewise 100 per cent equals eight times the volume, etc.

FIG. 1. Normal ovaries of a mare, typical of the first few days of heat. The ventral borders are towards each other, giving a view of the medial surfaces. The right ovary is comparatively quiescent, but contains a recent corpus luteum, 20 mm. x 12 mm., and a follicle 12 mm. in diameter. The left ovary is active, containing 7 palpable follicles ranging from 15 mm. to 48 mm. in diameter. Right ovary is 55 mm. long x 35 mm. wide x 40 mm. deep; weight about 50 grams. Left ovary is 90 mm. long x 45 mm. wide x 68 mm. deep; weight about 170 grams. (Actual size)

follicles, which may be from 4 to 7 cm. in diameter; (b) partly to smaller follicles (one to five are usually present; they usually vary from 1 cm. to 3 cm. in diameter) and (c) to a congestion and infiltration which noticeably enlarges and softens the stroma of the ovary.

The greatest reduction in size usually occurs near the end of heat in the ovary which has not just ovulated. This is due to the absence of a recent corpus luteum, to the reduction in size of most of the follicles and to the absence of any noticeable congestion. Mare XXV provides a good illustration of these

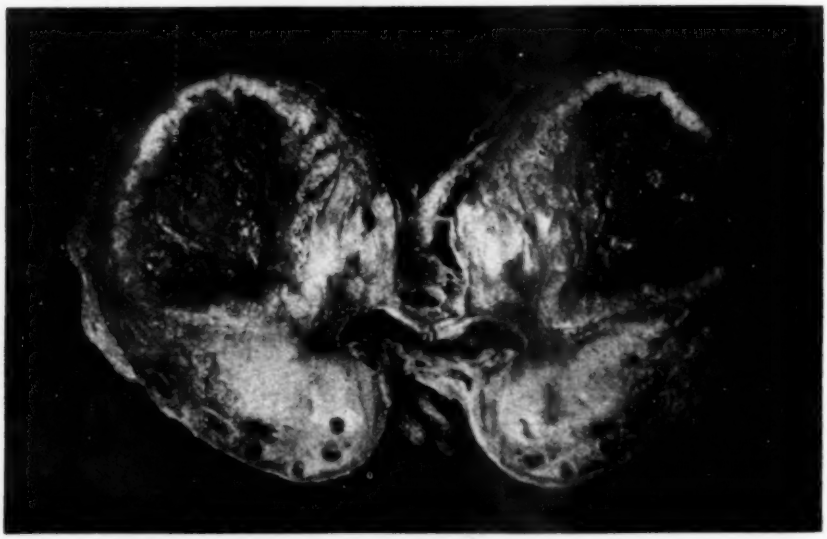

FIG. 2. Left ovary of a mare showing a recent corpus luteum in the anterior pole. The corpus is 35 mm. in diameter, with a point extending down to the ovulation fossa. The corpus shows the typical outer layer of folded, yellow, luteal tissue and the central, dark, gelatinous area. Ovarian stroma is shown below and behind it. Ovary is 55 mm. long x 45 mm. wide x 42 mm. deep; weight about 65 grams. (½ actual size)

remarkable changes: On June 29, the day before ovulation, her left ovary would probably have weighed about 300 to 325 grams, while the right ovary would probably have weighed only about 48 to 52 grams, a ratio of 6 to 1. Just before the next ovulation, 24 days later, the condition was reversed, the right ovary was about 280 to 300 grams and the left only about 40 to 45 grams, or a ratio of about 7 to 1, this time in favor of the opposite ovary.

The changes in a single cycle are not quite so radical when one ovulation succeeds another in the same ovary, since the presence of the corpus luteum prevents reduction to the minimum size. However, mare XXV again furnishes an instance of

marked variation. On May 12, shortly before ovulating, the right ovary, with a 6.5-cm. follicle and two others less than 1.5 cm., would have weighed about 200 to 225 grams; eight days later, with a corpus luteum about 3.5 cm. x 2.8 cm., it was estimated to weigh 50 to 55 grams; and June 7, with a 6-cm. follicle ready to rupture and one small follicle, it was again about 200 to 225 grams. This was a ratio of about 4 to 1 to 4.

As many as 15 new follicles may appear during one cycle,* the average being about 8 to 9. They may appear at any time during the cycle but their development seems to be stimulated in some way during the four or five days before and the first few days of heat. (It should be noted that this is coincident with the rapid reduction of the corpus luteum.) These follicles develop quite uniformly in both ovaries up to 2 or 3 cm. in diameter, but few, except those which are to rupture, ever get larger than 3 or 3.5 cm. In non-pregnant animals follicles which do not rupture may remain palpable from 1 to 35 days or more, averaging 7 or 8 days. Their maximum size varies from a mere soft spot to a diameter of 4½ to 5 cm. and is estimated to average about 2 or 2½ cm. Some large follicles develop early in the cycle. Occasionally one of these disappears suddenly, before the onset of heat, causing one to wonder if ovulation has occurred; another one may continue through heat and rupture at ovulation time; but usually they remain for several days, then gradually recede.

It would be interesting to know just how the follicles which rupture at ovulation time are selected. Some have been palpable for only five days, others as long as seventeen days before rupturing. They sometimes have developed a significant size as early as dioestrum and apparently dominate the field. In other cases they first appear during oestrus, sometimes after other follicles have attained a size of 3 to 5 cm., they then rapidly increase while their predecessors reduce. Occasionally no follicles over 2 cm. in diameter are present at the onset of heat.

Some mature follicles are very tense as ovulation approaches, but others are decidedly slack, so it seems that internal follicular pressure can have very little to do with causing ovulation.

Since ovulation always occurs through the ovulation fossa, the wall of the follicle and capsule of the ovary are left intact. The empty follicle then usually feels like a sac containing a small quantity of fluid. This content seems to increase in amount

*In the ovaries of mare II at least 33 follicles were present, none over 20 mm. in diameter.

and decrease in fluidity from day to day, while the capsule seems to shrink to fit it. The corpus luteum has usually attained its greatest size by the second to the fourth day. At this stage it may feel like a gelatinous body or it may fluctuate like a heavy fluid. In the latter case it might be confused with a follicle, particularly an atretic follicle, but it can be distinguished by its heavier fluid, thicker wall and less spherical shape.

The character of a mare's corpus luteum seems to change gradually but quite constantly. As it becomes older it usually becomes firmer but sometimes the reverse is true and a corpus which had been fairly firm may fluctuate.* The texture of some may be described as firm but elastic, while others seem rough, like a mass of angle worms under a membrane. The corpus is usually more or less pliable until so reduced it becomes lost to palpation.

Some of the reasons for the variance in character of different corpora lutea and for the changes noted are evident when ovaries with corpora lutea of different known ages are sectioned and examined. Briefly stated, the gross changes appear to be as follows:

Shortly after ovulation the cavity partially refills with a lymph-like fluid which usually has considerable hemorrhage in it. This fluid is surrounded by the developing luteal tissue, which forms a capsule or cortex for the developing corpus. This luteal tissue or cortex is a light-yellow color, at first very thin, but as it develops it is thrown into complicated folds which encroach upon the coagulated central mass. The latter develops a homogeneous, gelatinous appearance and is gradually resorbed until at about the next oestrus it is reduced to a streak which resembles a core in the center of the corpus. This core persists for several months or until the corpus is too small for the details to be recognized with the naked eye. The corpus luteum usually takes the shape of a pear or a toad-stool, with the convex surface against the capsule of the ovary and the stem extending to the fossa.

Thus the fluctuation often felt in a corpus luteum is due to the fluid which at first fills the cavity; the elastic feeling is caused by this fluid becoming coagulated and organized and also by the encroachment upon it of the luteal tissue; the rough, worm-like feeling is due to the foldings in the luteal tissue; and

*See the report on the corpus luteum of pregnancy in mare XXIII.

the progressing firmness is the result of absorption of the luteal tissue and the development of connective tissue in its place.

The maximum palpable dimensions attained by the corpus luteum are usually about one-half to three-fourths of the maximum dimensions of the follicle it replaced. Its volume is therefore probably only about one-sixth to one-half that of the follicle. Its shape depends upon the position and surroundings. It may extend from the end of the ovary like a disk-shaped rudder, it may form a longitudinal ridge along the sides or upper border of the ovary, or it may be quite spherical regardless of its position. A noticeable reduction in size usually commences anywhere from the sixth to twelfth day and continues until the corpus luteum can no longer be distingusihed. This occurs usually during the first part of the next heat period.

The corpus luteum of pregnancy develops just like the corpus luteum of oestrus and can not be distinguished from it, except by its length of life. Instead of reducing rapidly, in preparation for another heat period, it is usually easily palpated for several weeks.

We have followed four mares through the first twelve weeks or more of pregnancy. Two of these had double ovulations, so actually six corpora lutea were followed. Mare XXVI had two in her left ovary. One gradually reduced from about 4 cm. diameter on the fourth day, then 3 cm. on the fifteenth day, to 2 cm. on the thirtieth day, after which it could no longer be distinguished from ovarian stroma. The other corpus luteum reduced likewise but was lost among several developing follicles about the thirtieth day. When these follicles subsided, all traces of the corpus luteum had disappeared.

Mare XVIII had one corpus in each ovary. They likewise could not be distinguished after the fifty-seventh day. Mare XIV has been pregnant four months. Her corpus luteum reduced from about 3.5 cm. in diameter to 2.5 cm. by the thirtieth day, then remained until it is now about 1.7 cm. in diameter and is losing its characteristic texture.

Mare XXIII has been pregnant twelve weeks and her corpus is still prominent. However her case is very peculiar. The corpus reduced in a manner typical of a corpus luteum of oestrus, and although its site is one readily palpated, no trace of it could be palpated after the fifteenth day. However, on the thirty-ninth day the region was noticed to be softer and since then it

has gradually redeveloped until now it is nearly its original size.

We are anxious to know what the fate of the corpus luteum of pregnancy, in the mare, really is, but as yet have been unable to secure the necessary specimens with which to find out. In all other species of mammals, studied and reported, it remains prominent until about the time of parturition but we have never found a mare pregnant six months or more in which it could be palpated. In ovaries taken from mares near the end of pregnancy no recent corpora lutea were present.

Another condition peculiar to the mare is the development of many large follicles during the first few months of gestation. Often several follicles from 4 to 7 cm. in diameter will be present in one ovary at the same time, causing the ovary to assume a remarkable size, usually larger than it had ever been found before pregnancy. The left ovary of mare XXVI, on her fifty-seventh day of pregnancy, was the largest ovary yet palpated. It was estimated at 4¾ inches long, 3 inches wide and 3½ inches deep and would have weighed about 450 to 500 grams. It consisted of four or more follicles of 2.5 cm. to 7.5 cm. in diameter. It was almost oval and quite soft. The follicles which develop during pregnancy remain longer than usual, but never rupture.

After a few weeks of such activity the ovaries gradually reduce in size, almost cease forming follicles and after the fourth or fifth month go to the opposite extreme. They then become smaller than usual, very firm, and the poles are tapered in a peculiar manner. At this stage they are drawn down to, or below, the level of the pubis and held, in the border of the broad ligament, with the fossa facing forward. Towards the end of pregnancy they can scarcely be reached.

The changes in the uterus and other organs during pregnancy will not be discussed except to state that a positive diagnosis of pregnancy can be made, by finding a bladder-like dilatation in the uterus, occasionally as early as the twenty-ninth day but sometimes not until about the fiftieth day.

In conclusion we wish to emphasize the peculiarities of the reproductive phenomena of the ovary of the mare, for contrast with those of other species which have been studied. Briefly they are:

1. The ovary of the mare is relatively large and extremely variable in size.

2. *It has an ovulation fossa—a depression on the free (ventral) border—through which all follicles rupture.*
3. Germinal epithelium is confined to this fossa, the balance of the ovary being covered by peritoneum, under which is a strong fibrous capsule.
4. *Thr mature follicles attain a remarkable size and are often mistaken for and treated as cysts.*
5. Heat may last from four to eleven days, *but ovulation usually occurs during the last or next to the last day of heat.*
6. The corpus luteum never becomes nearly so large as the follicle it replaces.
8. The corpus luteum varies in consistency from firm to fluctuating.
8. It reaches its maximum size the second to fourth day and soon starts reducing slowly.
9. It is seldom palpable after the following heat period.
10. *The corpus luteum of pregnancy reduces, beyond what would be considered functional size, before the middle of the gestation period.*
11. The ovary develops its maximum size during an orgy of follicle formation early in pregnancy, then goes to the opposite extreme, its minimum size, late in pregnancy.

These remarks are based entirely upon gross observations. Later we hope to complete them by the addition of findings from microscopical studies.

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## TYPHOID VACCINE PER OS

Protection against typhoid by swallowing vaccine instead of having it injected under the skin is being tried out experimentally at the bacteriological laboratories at the State College of Washington, according to a note in *Science*. This method has the advantage of making the victims less sick than the customary "shot in the arm," but the degree of immunity it confers, compared with the older way, is not yet absolutely determined. The rate at which the immunity is developed is also unknown for the present, but it is hoped that further research will clear up these points and indicate the practical value of mouth vaccination.

# INHERITED DEFECTS OF LIVE STOCK*

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Nowadays most structural defects of new-born animals are regarded as heritable. This view is the result of scientists not being content to accept what they saw but in wondering how it came about. In order to secure this information the family histories of defectives have been studied and selected matings have been made.

In this paper an attempt will be made to correlate the facts relative to the etiology and occurrence of inherited defects of farm animals. It is hoped that the contribution and the discussion to follow will not only result in the reporting of more cases and in the collection of more data, but will also be a means of helping the members of the veterinary profession to give their clients an authoritative opinion about, and scientific explanations for, the occurrence of inherited defects in live stock. Eventually, this should help to overthrow the many entirely absurd traditional beliefs now held, not only in regard to the inheritance of defects, but also relative to many other specific characters. We refer particularly to telegony, maternal impressions, and to most so-called cases of the transmission of acquired characters.

## WHAT HEREDITARY DEFECTS ARE

Hereditary defects in animals are either anatomic or physiologic alterations. They are manifested either as changes in appearance or in function of the parts involved. Defects of this kind are also known as malformations, imperfections, anomalies and abnormalities. Similar defects due to aberrant embryologic development also occur, but they may or may not be heritable. In order to determine whether a given defect is heritable, breeding tests must be made, or family histories must be secured.

Two distinct types of inherited defects are recognized, *viz.*, recessive and dominant defects. Since the recessive defects are

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the more important from the standpoint of the live stock breeder, for reasons to be explained later, they will be considered first.

## The Nature of Recessive Defects

While there is no apparent difference between the appearance of recessive and dominant defects in animals, there is a decided difference between them so far as their inheritance is concerned.

Most animals showing recessive defects are the progeny of apparently normal parents, each of which, however, carries the factor not only for the defect, but also the factor for the normal. Since the factor for normal in these animals is dominant, it predominates and obscures the factor for the recessive. As such parents carry the factor for normal, it is evident that some of their offspring would be normal and carry no factors for recessive defects. On the other hand, others of their offspring would be like themselves and carry factors for defects. Accordingly, when animals that have no recessive factors for defects, irrespective of their parentage, are mated, their offspring are always normal.

Some defective animals, however, are the progeny of an apparently normal parent on one side and an evidently defective parent on the other side. Other defective animals are the progeny of parents both of which are abnormal. In these premises all the offspring would be abnormal, *i. e.*, defective. The two latter are seldom seen in domestic live stock because practical breeders do not ordinarily mate known defective animals.

A good example of the usual type of recessive defect is congenital cataract of cattle which has been studied by Detlefsen and Yapp (1920). As it is customary to designate the recessive factor by small letters and the dominant factor by large letters, this method will be used in explaining how this recessive defect is inherited. The parents of these defective calves are apparently normal and each carries the normal factor N, as well as the recessive factor n, in each body cell, accordingly an individual of this type is represented by Nn. Each sperm cell and each egg cell, however, carries only one of the pair of factors, as reductional division occurs during their production. So, on the average, half of the reproductive cells carry N and half carry n. No single sperm or egg cell can carry more than one factor of the pair. When fertilization takes place, one factor from each parent comes together in the new individual. Accordingly, each new individual will have one of three combinations, *viz.*: NN, Nn,

or nn. On the average, due to chance combinations, these are in proportion of NN : 2Nn : nn. Owing to the fact that NN individuals cannot be distinguished from Nn individuals, the ratio observed is three normal to one defective. One-third of the individuals which appear normal can never produce defective offspring, regardless of what they are mated with. The other two-thirds will produce three normal to one defective when mated with individuals carrying the same combination of factors. This

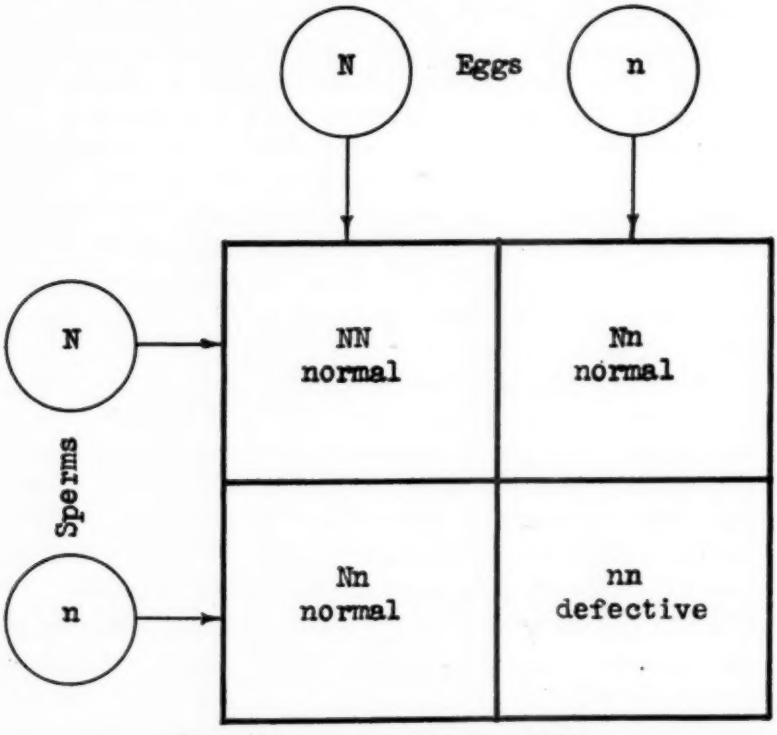


FIG. 1. INHERITANCE OF A RECESSIVE DEFECT

This checkerboard shows how congenital cataract of cattle, a recessive defect, is inherited. Both sire and dam appeared normal, yet both carry the factor for the defect as well as for normal. Consequently, out of four calves born from such a mating, the ratio would be one calf (NN) with neither cataract nor the recessive factor for it; two calves (Nn) apparently normal, yet carrying the recessive; one calf (nn) with cataract.

shows why the defect for cataract is carried in part of the stock, even though no animals with cataracts are used for breeding.

To help in understanding how congenital cataract of cattle is inherited, the checkerboard diagram in fig. 1 should be found of value.

## THE NATURE OF DOMINANT DEFECTS

As has been stated, it is impossible to distinguish by inspection between dominant and recessive defects. While both are due to

heritable factors, all animals showing dominant defects are the progeny of parents either one or both of which are similarly defective. In other words, dominant defects are due to factors which are carried only by abnormal appearing individuals.

A good example of a dominant defect in cattle is polydactylism, a condition studied and reported by Roberts (1921). The factor responsible for this defect may be designated by P and the factor for normal by p. As each individual carries two

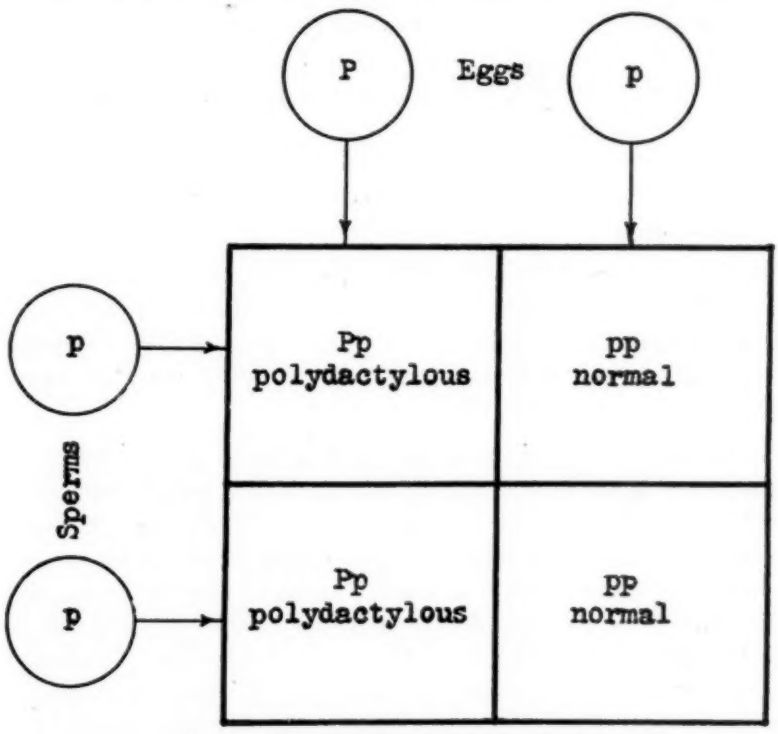


FIG. 2. INHERITANCE OF A DOMINANT DEFECT

The checkerboard above shows the results of mating a polydactylous cow with a normal bull. The defective animal, besides carrying the factor for extra toes (polydactylism), also carries the factor for normal. Out of the four calves from this mating, the ratio would be two polydactylous (Pp) to two normal (pp) calves. Or, in other words, equal numbers of the two kinds.

factors, the polydactylous individual may be represented by PP, or Pp; while the normal individual may be represented by pp. The checkerboard diagram in fig. 2 shows graphically the results of the mating mentioned above, *i. e.*, the ratio of polydactylous to normal calves would be 1 : 1, when a polydactylous bovine is mated with a normal bovine.

From what has been said it is evident that the important difference between a dominant and a recessive defect is that

normal parents mated together can never produce offspring with dominant defects, while apparently normal parents may produce offspring with recessive defects.

## Defects Known To Be Inherited

All types of domestic animals may have heritable defects. Economically, however, only those that handicap the animal to such an extent as to jeopardize its actual existence are of importance. The factors responsible for such defects may be either semi-lethal or lethal, depending upon the nature and extent of the damage. Semi-lethal factors are incapable of causing death directly, yet may cause it indirectly by handicapping the animal in different ways. Lethal factors, on the other hand, cause death directly. In general, the former become effective after birth, while the latter destroy life before birth, *i. e., in utero.*

In horses, a heritable defect that has been studied is a genetic form of sterility. Wriedt (1924b) observed this defect in the Danish white horse. According to him it is caused by the action of a lethal factor and results in failure of the mare to bear a living foal due to death of the embryo.

In cattle, among inherited defects of interest are the following: Two different dominant ear defects of cattle, noted respectively by Yamane (1915) and Lush (1922); the "bull-dog" fetal calf, a condition caused by retarded functioning of the pituitary, and thought by Crew (1923) to have a definite genetic basis; polydactylism in cattle, recorded by Roberts (1921), already referred to; the defects mentioned by Wriedt in Norwegian Telemarken calves (1924a); the congenital cataracts in cattle described by Detlefsen and Yapp (1920) to which reference has already been made; the congenital epithelial defects of calves studied by Hadley (1926), which will be described later.

In sheep, Ritzman (1916) reported a short-eared condition. Wriedt (1921) reported a similar, if not identical, defect of sheeps' ears.

In swine, a character of considerable economic importance, although not strictly lethal, is that responsible for inherited hernias studied by Warwick (1926) and also to be referred to more specifically in this paper. Another abnormality of considerable interest in swine is mule-footedness, which was studied by Detlefsen and Carmichael (1921). Wattles of swine, while

not strictly defects, are definitely inherited according to Kronacker (1924).

In dogs, an inherited form of deafness has been studied. It appears to be a simple recessive, as is also cleft-palate in dogs. The latter defect, however, is lethal, since the pups cannot suckle. Both the above are mentioned by Crew (1925). Wriedt (1924a) has described a semi-lethal factor in grey-mottled dogs, such as the Great Dane, Dachshund, and Norwegian Dunker Hound, which results in small eyes (microthalmia), defective pupil (coloboma) and glaucoma.

In goats, Lush (1926) has shown that wattles are definitely dominant characters, as they also are known to be in swine. Cryptorchidism has been proved to be heritable in goats by Wolf (1923).

### Defects Probably Inherited

Among the noteworthy defects of live stock which are very probably inherited may be mentioned a defect of hair and teeth of cattle reported by Cole (1919); also the exceedingly interesting defects of the genital organs in a group of heifers due to arrested development of the Müllerian ducts, recently described by Fincher and Williams (1926).

Cryptorchidism is likely heritable in other animals as well as in goats, but this has not been experimentally proved. Entropion, as observed in lambs, dogs, and horses, may be heritable, as it seems to occur with greater frequency in certain breeds of these species than in others.

Many other defects, particularly those occurring in horses, are thought by some to be inherited, but more careful and extensive study must be made to determine this point. Among these so-called hereditary unsoundnesses are the following: amaurosis, bog spavin, cataract, melanosis, navicular disease, paralysis of left recurrent nerve (roaring), periodic ophthalmia (moon blindness), pulmonary emphysema (heaves), ringbone, sidebone, roach-back, sway-back, defects of the jaws resulting in undershot or overshot mouths, and congenital anomalies in which the teeth of the two arcades do not wear on each other.

While most defects of new-born animals are probably inherited, there is one interesting defect that strictly speaking is not. This is the abnormality observed in the genital organs of the so-called free-martin heifer, the co-twin to a bull, which renders her asexual. It is an acquired defect due to environmental influences

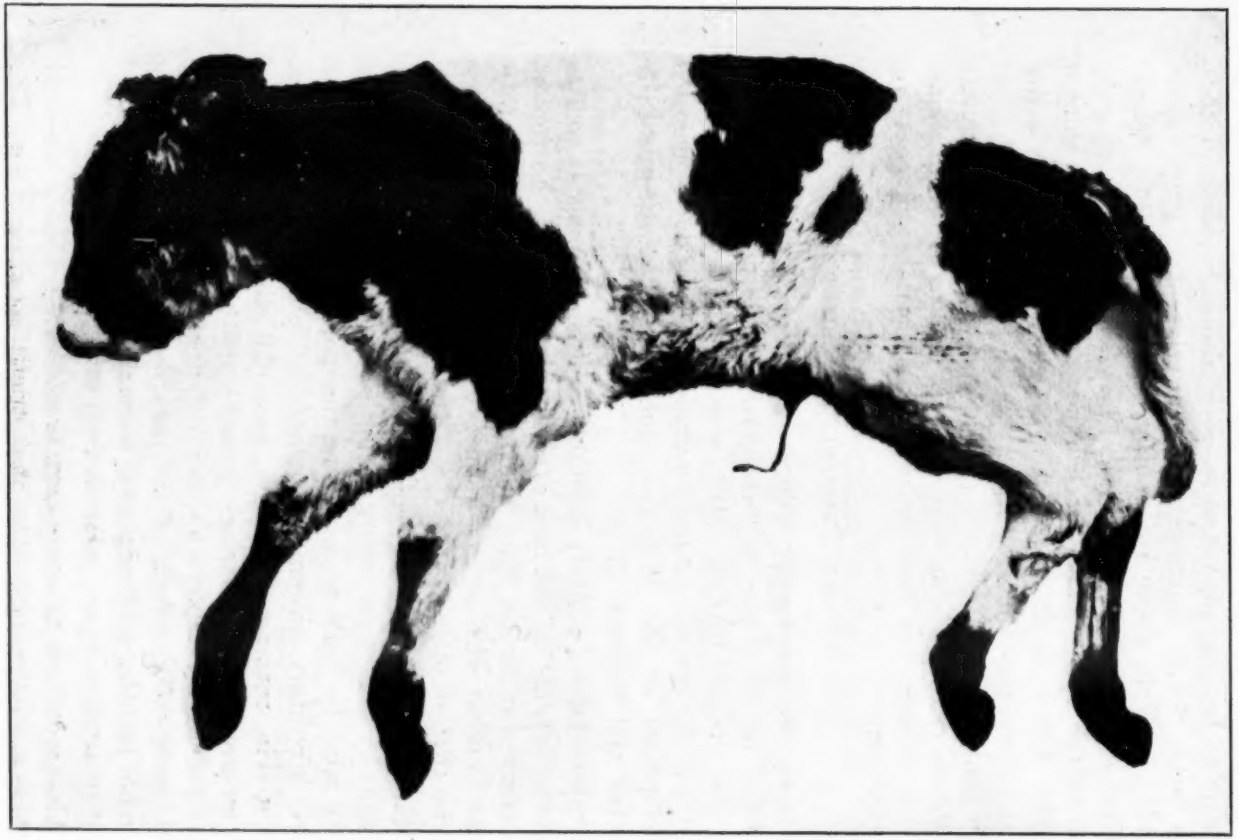

FIG. 3. CALF SHOWING CONGENITAL EPITHELIAL DEFECTS (Epitheliogenesis Imperfecta Neonatorum Bovis)

The lesions in this case were typical of the defect. Particular notice is drawn to the denuded and raw-appearing areas below both the knees and the hocks. The hoofs on the fore feet were normal, but no hoofs had developed on the hind feet. The ears were deformed, due to adhesions which took place between the defective epithelium before birth. The mucous membrance covering the muzzle and lining the nostrils was very thin and stripped off easily, leaving a raw surface. This calf was carried full term and weighed about 100 pounds.

occurring before birth. The cause seems to be an excess of the sex-hormone of the male twin over that of the female, as a result of an anastomosis forming between the blood-vessels of the two fetuses. Only about one free-martin in six becomes a breeder.

### Congenital Epithelial Defects of Calves

The senior author has identified and investigated this disease of new-born, full-term, normal-size calves. Briefly the lesions are as follows: defective formation of the skin below the knees and hocks; absence or incomplete development of one or more claws; deformed ears due to rolling of the margins and growing together of the contacted surfaces, which shows that these lesions antedated birth by some weeks; defects in the integument of the muzzle; defective areas in the mucous membranes of the nostrils, tongue, hard palate and cheeks. (See fig. 3.)

To date forty-three similarly defective calves have been detected. They were in thirteen different herds. Thirteen of the calves were from one herd where incestuous breeding had been practised. All herds were located in Wisconsin and were either pure-bred or grade Holsteins. The pedigrees of these calves have been carefully studied with the result that all have been found to trace to the same foundation stock that was imported from Holland about 1871. Moreover, similarly defective calves have been reported from Holland by Plank (1924) in herds carrying the same blood lines.

From the preliminary study, it is concluded that these inherited epithelial defects are the result of one pair of recessive factors. Both parents appeared perfectly normal, yet when mated produced distinctly abnormal, as well as normal, progeny. About one-fourth of the calves from these matings were defective. In other words, this is a recessive that shows itself in the calf only when both sire and dam carry the factors responsible for the defects. Although not strictly a lethal, it amounts to the same thing, as the defectives are never able to survive longer than a few weeks, due in part to septicemia developing from infection gaining entrance through the lesions.

### Inherited Hernias of Swine

The junior author has found that scrotal hernia of swine is definitely heritable. The defect is recessive, but not due to a single pair of recessive factors. However, it seems very probable that not more than two pairs of factors are involved. Three

generations of swine have been raised from herniated boars. In the first generation a herniated boar was mated to sows, most of which had definite relationships to herniated boars. Fifty-six boar pigs were raised to one month or more of age. Of these, eight individuals, or 14.28 per cent, had scrotal hernias. In the second generation only females from the first generation were bred to herniated boars. Fifty boar pigs were raised, of which twenty-one, or 42 per cent, had scrotal hernias. Sows from the second generation were bred to herniated boars and produced forty-four boar pigs. Nineteen of these, or 43.18 per cent, had scrotal hernias. Only one umbilical hernia appeared in the three generations.

The following two-factor hypothesis has been used to explain the inheritance: h and h′ are factors for inguinal hernia, both of which must be present in double or homozygous condition for hernia to be manifested in the boar pig. Herniated boars are, therefore, of the composition *hh h′h′*. Sows of the same genetic make-up are normal because of the anatomic differences associated with the female sex, inguinal hernias being limited almost exclusively to the male in this species. From this it follows that boars with scrotal hernias when bred to normal-appearing sows produce progeny all of which will be normal, or three-fourths of of the males will be normal and one-fourth herniated, or one-half of the males normal and one-half herniated, depending upon the genetic make-up of the sows. Certain boars may be normal, but when bred to certain sows part of the male progeny will be herniated. The boar pigs produced may be in the proportions of 15:1, 7:1, 3:1, or 1:1 normal to herniated, respectively, according to the factor complexes of the parents. Bred to sows of other genetic make-up the same boars would sire only normal offspring.

## How Defects Are Transmitted

It is obvious from what has been stated that appearance alone cannot be relied upon as a guide in breeding operations. Recessive factors for defects may be in the germ-plasm of normal-appearing animals. They may be carried on and on for many generations before appearing in double, or homozygous, combinations, which is necessary before the defects will show as visible lesions. Whether the defects will appear depends upon the chance factor make-up of the particular animals mated. On the other hand, a dominant defect occurs whenever the factor

is present in either single or double dose. Therefore, elimination of recessive defects by ordinary methods of selection is slow, while elimination of dominant defects by the same methods is rapid and certain.

The term reversion is commonly used by stockmen in referring to defective animals. Specifically it applies to the reappearance of some recessive character which has been carried in the germ plasm of the intervening generations, without showing. Mutations, or sports, on the other hand, are individuals having factors which have not been present in the stock before. The cause of the appearance of mutations is not known. If the mutations are recessive, these factors may be carried in the germ plasm for many generations before the character itself appears. Probably most, if not all, of the defects of live stock are being carried in this way, without any defectives ever having reproduced themselves.

### Inbreeding in Relation to Defects

Inbreeding of live stock has been held in both the highest and lowest repute of any breeding practice ever followed. Moreover, most of the greatest founders of the modern breeds of live stock used it successfully. Disaster, however, has been experienced by some breeders after only one or a few generations. Observations, based upon many species of plants and of the smaller forms of animals after many generations of inbreeding, lead geneticists to believe that inbreeding of itself does not produce deleterious results. Inbreeding simply brings together like factors more often than is the case in outbreeding. Whenever pairs of recessive factors for defects are brought together, an individual with a double recessive is produced. Many other recessive factors, which have to do with the well-being of the individual, are carried in single, or heterozygous, form in most normal outbred stock. As long as these animals are mated to non-related stock, the chances of many of the offspring carrying double, or homozygous, recessives are rather small. Yet these hidden deleterious factors are being disseminated in the normal offspring stock until many animals carry the factors. Then, when these animals are mated together, disaster follows, whether related or not.

Although it is impossible to distinguish by inspection between animals carrying these damaging factors and those that do not, inbreeding brings them out if they are present. Highly inbred animals, which are fairly vigorous, are more reliable breeders

than outbred animals of more vigor. This is because inbreeding has eliminated many of the deleterious recessive factors from the germ plasm. Inbreeding with intelligent selection has been likened to the surgeon's knife, which removes hidden lesions, so that what remains will be better. But, like the surgeon's knife, inbreeding may be expensive in that the unfavorable recessives may appear in so many of the offspring that the breeding stock is wiped out. This statement is supported by the adage, "The operation was a success, but the patient died." Considerable chance is taken in these premises, owing to the fact that there is no way of knowing whether any particular stock will survive inbreeding except by trial. So the question often resolves itself to one of whether the breeder can afford to take the necessary chances in order to improve the final product.

## How To Avoid the Occurrence of Defects

Animals showing defects are usually discarded as a matter of course without their having an opportunity to propagate. However, this is not enough, as may be seen in many herds of swine where no boars with scrotal hernia have ever been used, yet in which herniated pigs appear. The practice of discarding the defective boars helps greatly in reducing the percentage of herniated animals in the herd. But to play safe the dams of defective offspring should also be discriminated against. This is because some of the brothers and sisters of defective stock will carry factors for the defect. On the other hand, some of them will probably be entirely free from the defect factors, but it would require expensive breeding tests to determine which ones these are. In certain herds it would also be well to discard all of the progeny of sires which have produced any defective offspring. In most herds this procedure would probably not be practicable.

It is realized that some of these suggestions may be quite drastic; yet when one appreciates the strong tendency for the defects to persist, they are reasonable. How rigid to make the selection depends upon the seriousness of the defects and the relative merit of the blood lines in other respects. Both of these facts must be considered by the breeder in making his decision.

Another matter of interest to veterinarians, in this connection, is the question of professional ethics. In discussing this subject, Fincher and Williams (1926) say: "One principle is clear; it is as much the duty of the veterinarian to the community and to

his state, to use whatever influence and power he possesses to prevent the production of anatomically defective animals, as it is to prevent the spread of infectious diseases."

In closing, it should be stated that even though some defects are not heritable, we believe it is wise to assume that any particular defect is heritable until proved otherwise. So veterinarians should advise their clients accordingly and tell them how to avoid the production of defective animals. Inbreeding should not be condemned as the cause of the defects, but considered as the means of bringing to light the factors already present which are the cause. This advice is based upon scientifically demonstrated facts, for details of which reference should be made to textbooks on animal genetics.

This mention of animal genetics suggests the desirability of including a course in the science of animal breeding in the veterinary college curriculum, as it has already been in the agricultural college curriculum. At least two veterinary faculties (Kansas and Edinburgh) have already done this and now offer such a course. In the opinion of the writers this is a distinct advance in the teaching of veterinary medicine that may well be carefully considered by other faculties in their efforts to improve their curricula. If the young men who are graduated in veterinary medicine in the future do not have this knowledge, they cannot expect to advise with or retain the confidence of their clients who have had an opportunity to study animal genetics.

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## BUREAU TRANSFERS

Dr. Thomas M. Owen (Ont. '97), from Norfolk, Va., to Richmond, Va., on tuberculosis eradication.

Dr. Harry F. Kern (Colo. '11), from Washington, N. C., to Suffolk, Va.

Dr. Harry P. Waddle (K. C. V. C. '10), from Washington, N. C., to Suffolk, Va.

Dr. S. H. Still (K. C. V. C. '11), from Washington, N. C., to Suffolk, Va., acting in charge.

Dr. Wm. J. Smith, from Chicago, Ill., to Kansas City, Kans., on virus-serum control.

Dr. Asa R. Andrews, from Chicago, Ill., to Portland, Ore., on meat inspection.

Dr. LeRoy P. McArdle (K. C. V. C. '13), from Omaha, Nebr., to Watertown, So. Dak., in charge of meat inspection.

Dr. Wm. D. Fountain (Colo. '24), from Omaha, Nebr., to Denver, Colo., on meat inspection.

Dr. Samuel D. Bamber (Cin. '17), from Madison, Wis., to Albany, N. Y., on tuberculosis eradication.

Dr. Benj. T. Woodward (U. P. '02), from Washington, D. C., to New York, N. Y., in charge of inspection and quarantine.

Dr. Daniel J. Bynacker (U. S. C. V. S. '13), from Columbia, S. C., to Jackson, Miss., on tick eradication.

Success means nothing more nor less than doing the work you are suited for, to the best of your ability. It means getting fitted into your proper place in life's machinery, doing each day's work honestly, living cleanly and thriftily, and trying each day to improve upon the work of the day before.

# SOME PHASES OF CANINE DISTEMPER*

*By* ASHE LOCKHART, *Kansas City, Mo.*

This paper is not offered as a complete treatise on canine distemper, but merely records some observations made by the writer and his associates, Dr. J. D. Ray and Dr. J. S. Barbee, in the course of several years' work with this disease.

While everyone interested in dogs is familiar with distemper, there are many confusing ideas concerning its pathology, and consequently standard literature contains many statements which serve only to befuddle our minds.

Early in our work we were impressed with the difficulty of preventing natural infection in experiment dogs. Regardless of the best quarantine we could establish, susceptible dogs developed distemper. We believe that this is in a measure responsible for the conflicting findings of investigators with reference to the cause of distemper. Some of our observations have direct bearing upon the work of small-animal practitioners, and it is with these things that this paper deals.

## TRANSMISSIBILITY

With the possible exception of foot-and-mouth disease, we do not believe there is a more highly transmissible disease affecting the lower animals. The enormous number of affected dogs should be sufficient indication of this fact, but if one is in doubt, try controlling distemper in a kennel by isolation, disinfection, etc. These measures are a great help, and should never be disregarded, but are less effective in dealing with distemper than in most other diseases.

## INCUBATION PERIOD

In various standard texts, the period of incubation of distemper is considered as being from two to fourteen days. A report of the Field Distemper Committee places it at four days. For several years we have had occasion to bring to our premises a large number of young dogs, thought to be susceptible to distemper. These dogs were obtained from rural districts and in nearly all cases there was a clear history of no exposure to distemper. On the same day that they were brought from the

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country they were exposed to distemper by being placed in contact with dogs sick of this disease.

During the past year we have handled 34 dogs in this way, the temperature readings and other observations having been made in conjunction with and under the supervision of the federal Bureau of Animal Industry. While these are not all of the dogs which have been handled in this way, we believe this group will suffice to show what happens when a susceptible dog is given a severe exposure.

These dogs were used to determine the susceptibility of larger groups of dogs, and were selected from the larger groups by a Bureau of Animal Industry representative. They were not given any kind of treatment, except for being dipped for fleas and lice, and in a few cases vermifuges were administered.

This group of dogs is selected for illustrating the things which happen when a susceptible dog is exposed to distemper, because they present practically the same phenomena observed in many others, and because the observations on these were made not only by ourselves, but also by a representative of the federal Bureau of Animal Industry.

The following table gives the date of exposure, daily temperature record, time after exposure when first visible symptoms appeared, time when a positive diagnosis of distemper was made, and outcome.

By careful study of this table it will be seen that, of the thirty-four dogs presented, one remained healthy, one was only slightly sick, one was healthy through the ordinary observation period but developed distemper later, while thirty-one developed the disease, showing a high percentage of susceptibility. The mortality rate indicates an intense virulence of the infection.

It is seen that several days after exposure the dog's temperature rises one or more degrees F., usually recedes for a few days, recurs in four or five days from the original elevation and, in cases which live for some time, varies considerably after the second elevation. It is also seen that almost without exception no ordinary symptoms, such as nasal or ocular discharge, sneezing, coughing, etc., were observed until after the second rise of temperature. The shortest period of time after exposure at which visible symptoms appeared was nine days. Of the thirty-one typical cases the average period which elapsed after exposure was 13½ days, while 76+ per cent showed first visible symptoms from the twelfth to fifteenth days.

A point of interest to practitioners is that, between the first and second elevations of temperature, the dog is usually practically normal, and may readily be thought to be a healthy dog.

While not brought out in the table, it was observed that parasitism, unthriftiness, overcrowding or other abnormal conditions shortened the period of incubation.

### Symptoms

The accompanying table emphasizes some things about the development of distemper symptoms which we believe are of marked significance. The most constant symptom of distemper which we have observed is the double rise of temperature, beginning usually between the sixth and eleventh days after exposure and accompanied by rapid loss of weight. This is followed by the development of other symptoms, such as sneezing, coughing, nasal and ocular discharge, dysentery or diarrhea, skin pustules and nervous derangements. We have not observed skin pustules or nervous derangements in any dog until at least twenty-one days after exposure.

### Lesions

The lesions found in the various stages and so-called types of distemper are interesting. If a dog dies or is destroyed during the early stages, before complications arise, few lesions are present. Petechial hemorrhages are sometimes found on the heart, pleura, peritoneum and kidneys; the carcass is apt to be somewhat emaciated and the various tissues may present a parboiled appearance.

If complications have set in, the lesions of course will vary with the locatiou of the disturbance. In respiratory cases, inflammation of the respiratory mucosa and, in many cases, purulent, catarrhal pneumonia, are found. Meningitis or an excessive amount of cerebral fluids is found in the nervous complications. Inflammation and hemorrhage of the intestinal or gastric mucosa accompanies enteric complications, and characteristic pustules are seen in skin cases.

### Complications and Bacteriologic Findings

Observations like those noted have led us to believe that true distemper is a systemic disease, and that the things which are usually considered visible symptoms are secondary in character, being produced by organisms which are ordinarily of low virulence but are capable of producing distrubances in devitalized

TABLE I—*Clinical records of*

| Dog | Date Exposed | Temperature 1st Day After Exposure | 2nd Day | 3rd Day | 4th Day | 5th Day | 6th Day | 7th Day | 8th Day | 9th Day | 10th Day | 11th Day | 12th Day | 13th Day | 14th Day | 15th Day | 16th Day | 17th Day | 18th Day | 19th Day | 20th Day | 21st Day | 22nd Day | 23rd Day | 24th Day | 25th Day | 26th Day |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 303 | 8-6-25 | | 2.4 | | 2.4 | 1.8 | 1.0 | 1.2 | 1.0 | 2.2 | | 4.0 | Di | ed | | | | | | | | | | | | | |
| 307 | " | | 1.4 | | 2.0 | 1.8 | 1.8 | 2.0 | 2.4 | 3.6 | | 2.8 | 2.6 | 2.0 | 3.6 | 2.2 | 4.0 | | 3.6 | 3.8 | 4.0 | 3.6 | 4.2 | 3.8 | | 4.0 | 3.6 |
| 312 | " | | 1.8 | | 3.6 | 3.4 | 2.0 | 2.0 | 2.8 | 2.8 | | 4.6 | 3.2 | 2.8 | 4.0 | 5.4 | 4.0 | | 3.2 | Di | ed | | | | | | |
| 314 | " | | 1.0 | | 1.8 | 2.0 | 2.6 | 2.8 | 2.4 | 2.6 | | 3.0 | 4.0 | 4.0 | 3.6 | 2.4 | 2.8 | Di | ed | | | | | | | | |
| 317 | " | | 1.8 | | 2.8 | 2.2 | 3.0 | 2.2 | 2.0 | 2.0 | | 3.0 | 4.0 | 4.2 | 3.6 | 4.0 | 3.8 | | 4.6 | 3.8 | 3.4 | 2.8 | 4.8 | 4.6 | | 5.4 | 5.0 |
| 319 | " | | 2.8 | | 2.2 | 3.0 | 4.0 | 2.2 | 2.0 | 2.0 | | 2.4 | 4.0 | 5.2 | 4.8 | Di | ed | | | | | | | | | | |
| 321 | " | | 2.8 | | 2.6 | 3.0 | 3.8 | 4.0 | 2.0 | 2.0 | | 4.8 | 3.0 | 4.0 | 4.2 | 5.4 | Di | ed | | | | | | | | | |
| 326 | " | | 1.4 | | 2.6 | 3.4 | 2.6 | 2.2 | 1.8 | 1.4 | Di | ed[2] | | | | | | | | | | | | | | | |
| 330 | 8-8-25 | | 1.0 | 1.6 | 1.0 | 1.0 | 1.6 | 2.0 | | 1.8 | 2.0 | 2.0 | 2.0 | 2.8 | 2.8 | | 4.4 | 3.8 | 4.0 | 4.0 | 4.6 | 5.2 | | 5.6 | 4.6 | Di | ed |
| 335 | 8-11-25 | 2.4 | 2.0 | 1.8 | 2.0 | | 2.0 | 2.0 | 2.0 | 2.0 | 3.2 | 2.0 | Di | ed | | | | | | | | | | | | | |
| 575 | 11-23-25 | 1.6 | 1.8 | | 1.0 | 1.4 | | 3.6 | 3.6 | 2.8 | 1.6 | 3.0 | 2.8 | | 2.2 | 3.0 | 1.4 | 3.6 | De | str | oy | ed[1] | | | | | |
| 577 | " | 1.8 | 2.0 | | 1.0 | 2.0 | | 4.0 | 3.8 | 3.8 | 1.0 | 2.4 | 2.0 | | 2.4 | 2.0 | 2.0 | 2.2 | 2.6 | 1.0 | | 1.6 | 2.8 | 2.0 | 1.2 | 2.0 | 1.0 |
| 582 | " | 1.8 | 2.2 | | 0.4 | 1.0 | | 5.0 | 4.6 | 3.2 | 1.0 | 2.4 | 1.8 | | 1.4 | 2.6 | 1.4 | 2.0 | 2.6 | 0.0 | De | str | oy | ed[1] | | | |
| 585 | " | 2.1 | 2.0 | | 1.0 | 1.8 | | 3.4 | 2.0 | 2.0 | 2.0 | 2.2 | 1.4 | | 2.0 | 2.0 | 2.0 | 1.6 | 2.8 | 2.0 | | 3.2 | 1.4 | 2.4 | 3.8 | 4.2 | De |
| 586 | " | 1.8 | 1.4 | | 1.8 | 2.6 | | 5.0 | 2.4 | 2.6 | 2.0 | 3.0 | 3.4 | | 2.2 | 2.0 | 3.0 | 2.0 | 2.0 | 3.0 | | 3.6 | De | str | oy | ed[1] | |
| 588 | " | 1.8 | 1.6 | | 0.6 | 2.0 | | 1.4 | 2.2 | 2.0 | 1.8 | 2.0 | 2.4 | | 3.2 | 4.0 | De | str | oy | ed[1,3] | | | | | | | |
| 593 | 11-27-25 | 2.0 | | 2.4 | 2.6 | 2.6 | 2.2 | 2.0 | 2.4 | | 2.4 | 2.0 | 2.4 | 3.0 | 2.0 | 1.6 | | 3.4 | 2.0 | 2.0 | 2.0 | 2.2 | 2.6 | | 2.6 | 2.4 | 2.0 |
| 597 | " | 2.0 | | 2.0 | 2.2 | 2.0 | 1.6 | 2.4 | 2.0 | | 2.4 | 3.0 | 2.6 | 2.0 | 3.0 | 4.0 | | 4.2 | 3.2 | 3.4 | 2.6 | 3.2 | 1.2 | Di | ed | | |
| 599 | " | 1.2 | | 2.0 | 2.4 | 2.6 | 3.0 | 4.2 | 3.4 | | 3.0 | 2.6 | 3.0 | 3.0 | 3.4 | 2.6 | | 4.0 | 5.0 | De | str | oy | ed[1] | | | | |
| 743 | 1-29-26 | 2.4 | | 2.2 | 2.0 | 2.0 | 2.0 | 2.2 | 2.4 | | 2.6 | 4.0 | 5.2 | 5.2 | 4.0 | 4.0 | | 4.0 | 4.6 | 5.4 | De | str | oy | ed[1] | | | |
| 744 | " | 1.6 | | 2.0 | 2.6 | 1.8 | 1.6 | 1.6 | 1.8 | | 4.0 | 3.4 | 4.0 | 4.6 | 3.4 | 3.6 | | 3.0 | 4.2 | 4.0 | 3.4 | De | str | oy | ed[1] | | |
| 746 | " | 3.6 | | 2.0 | 2.8 | 3.0 | 2.6 | 2.4 | 1.8 | | 1.2 | 2.0 | 1.8 | 2.0 | 2.8 | 2.0 | | 2.0 | 3.6 | 3.2 | 1.2 | 2.8 | 2.4 | | 2.0 | 2.4 | 3.0 |
| 856 | 3-27-26 | | 1.6 | 1.8 | 2.2 | 3.4 | 3.8 | 3.2 | | 4.0 | 4.0 | 4.2 | 4.4 | 1.2 | 2.6 | | 1.2 | 2.2 | 1.4 | 2.0 | 3.0 | 1.4 | | 1.0 | 2.6 | 0.0 | 3.0 |
| 857 | ' | | 2.0 | 1.0 | 2.4 | 3.4 | 3.2 | 2.8 | | 4.0 | 4.0 | 4.2 | 4.4 | 4.2 | 4.4 | | 3.2 | 5.2 | 3.6 | 4.6 | 5.0 | 4.0 | | 3.4 | Di | ed | |
| 858 | " | | 1.4 | 1.6 | 3.4 | 2.4 | 2.6 | 2.2 | | 3.0 | 3.8 | 3.0 | 4.8 | 4.0 | 4.6 | | 6.2 | 3.6 | 4.8 | 4.2 | 4.4 | 4.2 | Di | ed | | | |
| 867 | " | | 1.0 | 1.2 | 1.4 | 2.2 | 3.8 | 2.8 | | 3.2 | 2.8 | 3.0 | 3.4 | 2.0 | 2.2 | | 3.4 | 4.2 | 3.8 | 3.6 | 4.0 | 5.0 | | 2.8 | 3.6 | 3.0 | 3.8 |
| 870 | " | | 2.6 | 2.6 | 3.2 | 5.0 | 3.2 | 3.0 | | 3.4 | 3.8 | 4.0 | 4.8 | 3.6 | 3.0 | | 4.0 | 3.6 | 1.8 | 4.0 | 4.0 | 4.0 | | 4.0 | 4.0 | 3.6 | 4.0 |
| 871 | " | | 2.2 | 2.2 | 2.0 | 2.8 | 3.2 | 1.6 | | 2.0 | 2.0 | 2.0 | 2.0 | 3.2 | 3.0 | | 3.8 | 3.2 | 3.0 | 3.2 | 1.6 | 3.2 | | 2.0 | 1.0 | 1.4 | Di |
| 947 | 6-4-26 | 0.2 | | 1.0 | 1.6 | 1.0 | 1.8 | 2.0 | 2.4 | | 2.4 | 2.8 | 2.4 | 2.2 | 2.0 | 3.6 | | 3.4 | 4.0 | 5.2 | 4.0 | 3.2 | 3.6 | | 3.6 | 3.6 | 3.6 |
| 948 | " | 0.4 | | 1.6 | 1.0 | 1.2 | 1.6 | 2.4 | 2.2 | | 1.0 | 1.6 | 2.0 | 1.8 | 1.0 | 1.4 | | 2.2 | 2.4 | 2.4 | 2.0 | 2.2 | 1.6 | | 1.6 | 2.0 | 1.8 |
| 949 | " | 1.6 | | 1.6 | 1.8 | 1.4 | 1.6 | 2.2 | 3.6 | | 4.0 | 3.0 | 3.2 | 3.4 | 2.6 | 4.2 | | 3.2 | 4.0 | 3.6 | 3.0 | 2.4 | Di | ed | | | |
| 964 | 6-11-26 | 2.8 | | 2.4 | 1.6 | 1.8 | 2.2 | 1.0 | 3.0 | | 2.2 | 2.8 | 2.2 | 4.0 | 3.4 | 4.0 | | 4.6 | 4.2 | 4.0 | 4.2 | 4.0 | 4.2 | | 3.4 | 3.2 | 2.8 |
| 965 | " | 1.8 | | 1.6 | 1.4 | 2.0 | 1.6 | 1.8 | 3.0 | | 2.2 | 2.2 | 2.0 | 3.2 | 2.6 | 3.2 | | 4.0 | 4.0 | 3.8 | 4.0 | Di | ed | | | | |
| 972 | " | 2.4 | | 1.4 | 1.0 | 1.2 | 2.0 | 2.0 | 3.2 | | 2.0 | 3.0 | 3.2 | 3.0 | 3.6 | 3.6 | | 4.0 | 4.4 | 4.4 | 4.4 | 5.0 | Di | ed | | | |

[1]Dogs 575, 582, 585, 586, 588, 599, 743, and 744 were destroyed when it became clear that they were hopelessly sick.

[2]Dog 326 had a heavy hookworm infestation,

[3]Dog 588 had a heavy parasitic infestation.

*dogs used in making observations*

| 27TH DAY | 28TH DAY | 29TH DAY | 30TH DAY | 31ST DAY | 32ND DAY | 33RD DAY | 34TH DAY | 35TH DAY | 36TH DAY | 37TH DAY | 38TH DAY | 39TH DAY | 40TH DAY | 41ST DAY | 42ND DAY | 43RD DAY | 44TH DAY | 45TH DAY | 46TH DAY | 47TH DAY | 48TH DAY | 49TH DAY | 50TH DAY | 52ND DAY | DAY OF TEST ON WHICH FIRST VISIBLE SYMPTOMS WERE OBSERVED | DAY ON WHICH DIAGNOSIS OF DISTEMPER WAS MADE. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | | | | | | | | | | | | | | | | 11 | 12 |
| 3.8 | 3.8 | 3.8 | 3.6 | | | 4.0 | 4.2 | 4.6 | 6.0 | 2.2 | Di | ed | | | | | | | | | | | | | 13 | 13 |
| | | | | | | | | | | | | | | | | | | | | | | | | | 11 | 13 |
| | | | | | | | | | | | | | | | | | | | | | | | | | 12 | 13 |
| | Di | ed | | | | | | | | | | | | | | | | | | | | | | | 12 | 13 |
| | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | | | 12 | 13 |
| | | | | | | | | | | | | | | | | | | | | | | | | | 13 | 13 |
| | | | | | | | | | | | | | | | | | | | | | | | | | 9 | ---- |
| | | | | | | | | | | | | | | | | | | | | | | | | | 10 | 13 |
| | | | | | | | | | | | | | | | | | | | | | | | | | 10 | 10 |
| | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | | | 15 | 15 |
| | 2.6 | 0.0 | 1.6 | 3.0 | 98. | 1.0 | Di | ed | | | | | | | | | | | | | | | | | 15 | 18 |
| | | | | | | | | | | | | | | | | | | | | | | | | | 15 | 17 |
| str | oy | ed[1] | | | | | | | | | | | | | | | | | | | | | | | 16 | 19 |
| | | | | | | | | | | | | | | | | | | | | | | | | | 12 | 16 |
| | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | | | 14 | 14 |
| | 2.2 | | 2.0 | 1.6 | 2.2 | 2.6 | 1.4 | | | 1.6 | 1.8 | 2.2 | 2.2 | 3.2 | 2.0 | | 2.6 | 2.4 | 1.6 | 2.0 | 1.6 | | 2.2 | 3.0 | 58 | 62 |
| | | | | | | | | | | | | | | | | | | | | | | | | | 17 | 17 |
| | | | | | | | | | | | | | | | | | | | | | | | | | 12 | 17 |
| | | | | | | | | | | | | | | | | | | | | | | | | | 14 | 18 |
| | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | | | 14 | 18 |
| 2.4 | 3.6 | 3.2 | | 3.0 | 2.0 | 1.6 | 2.6 | 2.2 | 2.6 | | 3.6 | 1.0 | 2.4 | 2.6 | 2.8 | 3.2 | | 2.8 | 1.8 | 2.4 | 1.8 | 2.4 | 2.4 | 2.0 | 19 | ----[4] |
| 2.0 | 0.0 | | 1.2 | 1.6 | Di | ed | | | | | | | | | | | | | | | | | | | 15 | 16 |
| | | | | | | | | | | | | | | | | | | | | | | | | | 14 | 17 |
| | | | | | | | | | | | | | | | | | | | | | | | | | 14 | 16 |
| | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | | | 13 | 14 |
| 4.2 | 3.2 | | 1.8 | 3.0 | 2.6 | 3.0 | Di | ed | | | | | | | | | | | | | | | | | 13 | 13 |
| 3.6 | 1.4 | Die | d | | | | | | | | | | | | | | | | | | | | | | 13 | 13 |
| ed | | | | | | | | | | | | | | | | | | | | | | | | | 18 | 18 |
| 3.6 | 4.0 | Die | d | | | | | | | | | | | | | | | | | | | | | | ---- | ----[5] |
| 2.4 | 1.6 | 2.2 | | 2.0 | 2.0 | 1.4 | 2.0 | 1.6 | 1.8 | | 2.0 | 1.8 | 2.0 | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | | | | | | | | 14 | 14 |
| 2.2 | Di | ed | | | | | | | | | | | | | | | | | | | | | | | 13 | 13 |
| | | | | | | | | | | | | | | | | | | | | | | | | | 14 | 17 |
| | | | | | | | | | | | | | | | | | | | | | | | | | 13 | 15 |

[1]Dog 746 did not have typical distemper.
[2]Dog 948 remained healthy, except for injuries received in a fight.

tissues. If this be true, it would explain the well-known phenomenon of immunity to the various types of distemper, in a dog which has recovered from one type.

The bacterial organisms recoverable from distemper cases vary greatly, but in a general way are correlated with the symptoms and lesions presented. When respiratory disturbances are present *Alcaligenes bronchisepticus*, staphylococci and streptococci may be found, and in many cases of extensive pnemonia the *Alcaligenes bronchisepticus* is found in practically pure culture. An organism belonging to the Salmonella group is found in the so-called enteric type of distemper, and this organism in heavy dosage is capable of producing similar symptoms and lesions in old dogs known to have recovered from distemper.

In the type of distemper characterized by nervous derangements we have encountered an unusual type of organism from a high percentage of cases. This organism is found in the cerebral and spinal fluids. It is difficult to culture, making a sparse growth and dying quickly on ordinary media. While we have not yet been able to demonstrate compliance with all of Koch's postulates, the regularity of its occurrence, in those cases showing nervous derangement, and its absence in other cases points to its significance.

Our work with distemper has been done over a period of several years, and has required careful study of many hundreds of dogs. The work is being continued and it is hoped that more will be learned concerning the relationship of the various so-called types of distemper.

## Summary

1. A record of observations on the development of distemper in young dogs is presented.

2. It has been observed that there is usually a characteristic systemic reaction following distemper infection, this reaction being evidenced by a double elevation of temperature, the elevations occurring four or five days apart, and being associated with rapid loss of condition.

3. The first appearance of the manifestations of the so-called types of distemper usually do not occur until several days after the second elevation of temperature.

4. It is believed that the so-called types of distemper are due to secondary infections.

5. This work is being continued.

### Discussion

Dr. G. W. Little: There is no subject more interesting to this section of our profession than distemper, and I really believe that Dr. Lockhart and his associates are making an honest effort to solve some of the problems concerning this disease. In fact, a research along the lines of the causative agent of distemper should be stimulated in every possible way. The economic value of dogs in this country is tremendous. Sentimental value is just as tremendous as the economic value and therefore a research fund for the purpose of investigation of the causative factor in distemper could be raised easily and this fund administered by one of our colleges, and I am sure if such a fund was once started, and publicity given to it, we could have all the money necessary in a short time to carry out this work.

Now, then, as to the work of the Distemper Committee: They announce that there is an ultramicroscopic organism which has been found in ferrets and that ferrets are unusually susceptible to distemper. The professional pride of our bacteriologists is at stake as far as this disease is concerned. Why do they have to go to England, to Europe, to find the cause of distemper? We have a good many bacteriologists in America, and I feel that they are just as competent as any man over on the other side. If it is the lack of funds, I personally will do everything I can to stimulate this condition and try to get a fund started, and do everything in the way of publicity that I can to let my clients know that such a fund is started, and I am sure that many wealthy people, where the dog is really the child in the family, will come forward with hundreds of dollars for this fund and I propose that if this is possible that it should be administered by one of our colleges having bacteriologists competent for this work.

Now then, we come to the prevention of distemper. The elements entering into the prevention of distemper are various. First, in the young puppies just weaned, diet is a very important factor. Worm infestation is a very important factor—anything, in fact, that will reduce body resistance of the dog. And environment is an important factor. That is to say, dogs should be kept in sanitary quarters, with plenty of sunlight and air and given exercise. Not only that, but sporting dogs should not be kept in an apartment house, and no efforts upon the part of clients to breed dogs, such as the shepherd, in cities, in hotel rooms, which I have seen, should be encouraged.

Now then, when we think of Dr. Lockhart or Dr. Kinsley and his associates we think of Sero-Toxylin, and when this first came out I started to use it personally and, according to Kinsley's records, I have used between four and five hundred doses and from my experience I feel that this is of value. Any preventive serum is not infallible, by any means, and if one loses sight of the other factors in the prevention of disease, there is no agent for the prevention of distemper that will have much value. Any prophylactic agent is open to a great deal of criticism for the reason that people do not know how to use it. That is to say, in the case of Sero-Toxylin, a breeder will send his young dogs to the show, bring them home and then he remembers the danger of the contagion from distemper and thinks he will have Sero-Toxylin injected. This is the wrong idea, of course. Any preventive agent should be used purely as a preventive agent and any form of exposure should be anticipated before taking dogs to the show. Therefore, the failures and successes of Sero-Toxylin depend upon environment, diet and worm infestation, as well as carelessness in exposing the animal.

Now then, I am not going to say much more. I want to hear from a good many others on this subject, and especially Dr. Lockhart, and I hope he will bring out something in reference to Sero-Toxylin that we would like to know about.

Dr. Chas. W. Bower: I would like to ask about the use of Sero-Toxylin. For what length of time do you consider the immunity lasts? Also, is it safe and how long after inoculation is it safe to expose the animal to distemper? Thirdly, should not, even if the dog has received the inoculation, other precautions such as exposure to weather, be taken to get a safe protection.?

Dr. A. J. Steiner: If you have jurisdiction over a kennel, at what age would you vaccinate the pups? That might differ with the breed, but, I do not know. I would also like to ask Dr. Lockhart if he could say whether or

not that organism he has demonstrated in the spinal fluid, has ever been found within the brain?

DR. H. M. HAMILTON: After the dogs are inoculated, what chances of exposure have uninoculated or susceptible dogs during the period following inoculation?

DR. J. C. WRIGHT: Several doctors in the South, including Dr. Handley and several of the Carolina doctors and some from Alabama, have made complaint that Sero-Toxylin in some cases leaves a bad effect on the dog, one to three to five weeks after. The dog goes all to pieces and shows symptoms of chorea or locomotor ataxia. Some recover and do not develop distemper. On the directions they print never to split the dose. I followed those for a while and got some bad results on several cases. Since, I have split the dose on puppies and re-injected them with another half-dose, three weeks later, and on that I have never got a bad result. I have some of Dr. Cromwell's puppies now and I have injected them once and am going to inject them again when I get home.

CHAIRMAN FLYNN: You must bear in mind that Sero-Toxylin is not mentioned in the paper and Dr. Lockhart can use his own discretion in answering these questions.

DR. C. A. PLEUGER: If the toxin itself is injected, will it produce distemper without the use of the serum?

DR. LOCKHART: I very studiously avoided mentioning Sero-Toxylin in this paper. It seems, though, that many want it discussed. Dr. Bower asked concerning the duration of immunity. It is a little early to tell what the duration of the immunity may be following the use of this material. However, there are a great many dogs which have been treated for as long as two years and apparently they are still immune. They have been exposed to distemper but do not contract the disease. I think that depends largely, however, on the method of handling that particular dog. As to when it is safe to expose a dog, I will say that in our own work, practically all of the dogs which we have used have been exposed on the same day they were treated. I do not think that is a good procedure in general practice; the dog should be kept away from exposure as much as possible for a time, and it is certainly not a good procedure in your practice to invite disaster by placing a dog under adverse conditions following treatment. No matter what agent you may use, if you do not give the dog a chance, you will have more trouble than if you give him a reasonable chance.

Dr. Steiner asked concerning the age at which to vaccinate. In our work we have found that if a dog is given this material while, or shortly after, sucking an immune bitch, immunity in not obtained in many cases. Consequently, we advise that a dog be allowed to become at least three and preferably four months old before it is treated. One of the most successful users of Sero-Toxylin, Dr. Pfister, of Kansas City, in using this material, advises his clients to wait until the dogs are five or six months old. If they are treated younger, he gives it to them again and his results have been excellent.

With reference to the organism in the cerebral fluids, we have in some cases found that organism in the brain, but it is rather difficult to determine whether it is in the brain or in the fluids that are there. We find it in brain tissue proper, under certain conditions, particularly after the death of the dog. We found a little intra-cellular infiltration in those, but not a marked amount as a general rule.

Dr. Hamilton asks about vaccinated dogs transmitting the disease to others. I do not believe they will do it, although it would be bad practice, as a general rule, to treat one group of valuable puppies and leave the others untreated, because you never know when they will be exposed to distemper, and the owner may draw erroneous conclusions, should the untreated dogs contract the disease within a few weeks.

Dr. Wright brings up the size of doses, and says that some veterinarians have had trouble following the use of a full dose on very small dogs. We have heard very little about difficulties of this kind, and insofar as we know the percentage of such results is very small, otherwise it would, undoubtedly, have been reported to us. We would be glad to learn more about such cases, and hope that if any veterinarians meet with similar cases, they will report them.

However, I am firmly convinced that when nervous derangements occur in a relatively short time following the use of this material, the dog probably was in the incubative stage of distemper at the time of treatment. This is a difficult matter to determine. Distemper is so widespread that it can easily happen without your knowledge or the owner's.

With reference to the split dose, I will say that we have not been able to get good results with smaller doses than we recommend. We have used it on a number of young dogs, small puppies and the small breeds of dogs. We have not found, in our own work, that the dosage recommended produces a severe disturbance. We have not has good results where we reduced that dose.

DR. WRIGHT: Why could you not use that split dose and use it every two weeks?

DR. LOCKHART: I do not know. That is a question that can not be answered very well in view of our present knowledge. However, it might be similar to the case of using material on a puppy sucking an immune bitch. When a small degree of resistance is produced, the next injection does not produce a reaction. Answering Dr. Pleuger's question, as stated in the paper, everything that we used in trying to give a dog distemper, gave it to him. It is a difficult thing to keep a dog you do not want to get the disease from getting it, and every body who has worked with distemper has proved that everything he gives a dog produces distemper. For that reason we have not tried very hard to determine whether or not this antigen used alone will produce distemper. The dog will get distemper if you give it to him, but whether that is what gives it to him, I do not know.

DR. HAMLET MOORE: In answer to Dr. Wright's question on split doses, most of us are thoroughly acquainted with the physiological action of anything that is applied with therapeutic intent. A dose is a dose, while a half-dose is not a dose. Now there are a great many cases—everybody here has probably demonstrated that to themselves in using a drug for the first time—where, we will say, a grain was the prescribed amount, and was necessary to get the therapeutic value. Where a half-grain is given the therapeutic action is not obtained. I have seen that in a number of cases and in this case I do not know but what Dr. Lockhart fully explained that, that due to the resistance after the half-dose had been given, the second half rendered no resistance.

DR. C. C. RIFE: I would like to ask Dr. Lockhart about using the serum alone. In lots of cases coming to the hospital, where they have a high temperature, we think they are coming down with distemper. In those cases we have used serum alone and it seems that the period of distemper and severity of the disease is reduced. Do you attribute that to the use of the serum alone or what is it due to?

DR. LOCKHART: In our work with this material we have been impressed with the fact that the serum alone or serum in conjunction with the antigen has had little or no curative value, and while some veterinarians have reported very good results from the use of the material as a curative agent, using the serum alone or with the antigen, we do not feel that we can get good results and consequently I can not tell you much about it. We have not seen these good results, and I do not know to what to attribute them.

## INDIANA SHORT COURSE

Forty Hoosier veterinarians attended the special short course offered by Purdue University, at Lafayette, Ind., the week of November 1. Laboratory work, lectures and discussions covered a wide range of animal diseases, although poultry and cattle problems received most attention.

Do not concern yourself with what your friends say of you. Pay more attention to what your enemies say. They have an outside viewpoint.

# ANTHELMINTIC EFFICIENCY OF KAMALA AND TETRACHLORETHYLENE IN THE TREATMENT OF CHICKENS

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Although kamala has long been used as a teniacide in both man and animals, only very recently has the drug been recommended for this purpose in chickens. This recommendation by Hall and Shillinger[1] was made as a result of their work in which they found that an individual dose of one gram of the drug was sufficient to remove the tapeworms in about 95 per cent of the cases. Beach and Warren[2] have since obtained similar results.

Although kamala is primarily a teniacide, it will remove, according to Hall and Shillinger, from 5 to 10 per cent of the roundworms in chickens, but is not dependable for this purpose.

In previous experiments[3] the author showed that tetrachlorethylene was non-toxic for chickens except in extremely large doses and caused the removal of a large percentage of the roundworms. No teniacidal value whatever was noted.

With the anthelmintic properties of these two drugs in mind, it was thought that possibly the simultaneous administration of kamala and tetrachlorethylene might remove all of the tapeworms and roundworms when these parasites were present in chickens. Experiments were undertaken to determine the effect of the simultaneous administration of the two drugs, as well as the effect of kamala alone and the effect of kamala and tetrachlorethylene when given separately at intervals of three days.

All of the chickens used in these experiments were obtained from the local markets and, with the exception of eight broilers (2, 4, 10, 31, 41, 42, 13 and 43, table I), were adult birds classed as "culls." These latter birds were used because it was thought that, on account of their generally poor physical condition, in most instances, there would be a greater likelihood of their being infested with parasites.

In preparation for anthelmintic treatment the chickens were given their usual feed in the evening, after which all food remaining was removed. Treatment was given the following

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morning, food being withheld until about two hours after the administration of the anthelmintic. The diet of all consisted of cracked corn soaked with skim-milk. Kamala was given in the form of compressed tablets, each containing one gram. Tetrachlorethylene was given in soluble elastic globules of 1-cc capacity.

### Effect of Simultaneous Administration of Kamala and Tetrachlorethylene

In the tests to determine the effect of simultaneous administration of kamala and tetrachlorethylene, 27 chickens were used (table I). Their weights varied from 12 ounces to 6½ pounds.

Table I—*Simultaneous administration of 1 cc tetrachlorethylene and 1 gm. kamala. Autopsies in 2 to 32 days after treatment*

| Bird | Weight | Worms Passed | | Worms Postmortem | | |
|---|---|---|---|---|---|---|
| | | Round | Tape | Round | Tape | Cecal |
| 49 | 4 lb., 4 oz. | 1 | 2 | 0 | 0 | 0 |
| 38 | 3 lb., 6 oz. | 1 | 8 | 0 | 0 | 54 |
| 19 | 6 lb., 8 oz. | 0 | 8 | 2 | 0 | 62 |
| 33 | 2 lb., 10 oz. | 0 | 0 | 0 | 0 | 0 |
| 86 | 2 lb., 8 oz. | 0 | 0 | 0 | 5 | 0 |
| 29 | 3 lb., 4 oz. | 0 | 0 | 0 | 0 | 0 |
| 34 | 3 lb., 1 oz. | 0 | 0 | 0 | 0 | 0 |
| 24 | 3 lb., 6 oz. | 0 | 3 | 0 | 0 | 0 |
| 48 | 3 lb., 6 oz. | 0 | 0 | 0 | 0 | 0 |
| 15 | 3 lb., 5 oz. | 0 | 0 | 0 | 0 | 0 |
| 35 | 3 lb., 5 oz. | 0 | 4 | 2 | 0 | 0 |
| 85 | 2 lb., 11 oz. | 2 | 0 | 4 | 0 | 0 |
| 22 | 3 lb., 8 oz. | 2 | 0 | 5 | 12 | 0 |
| 16 | 4 lb. | 0 | 0 | 1 | 0 | 0 |
| 25 | 2 lb., 4 oz. | 0 | 2 | 0 | 0 | 0 |
| 28 | 3 lb., 12 oz. | 0 | 6 | 0 | 0 | 0 |
| 81 | 2 lb., 14 oz. | 0 | 0 | 0 | 0 | 0 |
| 45 | 2 lb., 14 oz. | 0 | 0 | 1 | 0 | 0 |
| 11 | 2 lb., 9 oz. | 0 | 1 | 0 | 4 | 0 |
| 2 | 1 lb., 6 oz. | 0 | 0 | 0 | 0 | 0 |
| 4 | 1 lb., 6 oz. | 0 | 0 | 0 | 0 | 0 |
| 10 | 1 lb., 4 oz. | 0 | 0 | 0 | 0 | 0 |
| 31 | 1 lb., 7 oz. | 0 | 0 | 0 | 0 | 0 |
| 41 | 1 lb. | 0 | 0 | 0 | 0 | 0 |
| 42 | 1 lb., 6 oz. | 0 | 0 | 0 | 0 | 0 |
| 13 | 1 lb., 5 oz. | 0 | 0 | 0 | 0 | 0 |
| 43 | 12 oz. | 0 | 0 | 0 | 0 | 4 |

These chickens were destroyed at intervals varying from 2 to 32 days after dosing. During this time they were held in individual cages, from which all droppings were removed daily and examined for parasites.

Postmortem examination of birds 25 and 33, which died 24 and 48 hours respectively after having received treatment, re-

vealed generalized tuberculosis but no parasites in the intestinal tract.

Chicken 10 died five days after treatment, postmortem showing coccidial infection. Chickens 43 and 42 died with similar postmortem lesions, in 24 and 48 hours respectively.

None of the other birds showed any abnormal symptoms during the time they were held, even though birds 29, 34, 15 and 28 were found on postmortem to have suffered from generalized tuberculosis. The internal organs of the remaining chickens of this group were apparently normal.

Of the 27 chickens in this group, 14 passed no parasites after treatment nor were any found on postmortem, 2 were infested with roundworms, tapeworms and cecal worms, 5 with roundworms and tapeworms, 2 with roundworms only and 4 with tapeworms only.

TABLE II—*Effect of kamala alone. Dose — 1 gm.*

| BIRD | WEIGHT | WORMS PASSED | | WORMS POSTMORTEM | | |
|---|---|---|---|---|---|---|
| | | ROUND | TAPE | ROUND | TAPE | CECAL |
| 30 | 4 lb., 8 oz. | 0 | 0 | 0 | 0 | 10 |
| 12 | 4 lb., 6 oz. | 0 | 2 | 0 | 0 | 0 |
| 27 | 2 lb., 4 oz. | 0 | 14 | 0 | 0 | 0 |
| 90 | 2 lb., 9 oz. | 0 | 0 | 0 | 0 | 0 |
| 1 | 3 lb. | 0 | 0 | 0 | 0 | 0 |
| 17 | 2 lb., 9 oz. | 0 | 1 | 0 | 0 | 0 |
| 37 | 2 lb., 13 oz. | 0 | 0 | 0 | 0 | 0 |
| 24 | 2 lb., 3 oz. | 0 | 0 | 0 | 0 | 0 |
| 91 | 2 lb., 15 oz. | 0 | 3 | 2 | 0 | 0 |
| 8 | 2 lb., 9 oz. | 0 | 1 | 0 | 0 | 0 |
| 29 | 3 lb., 2 oz. | 0 | 5 | 0 | 0 | 0 |
| 9 | 3 lb., 1 oz. | 0 | 0 | 0 | 0 | 0 |
| 82 | 1 lb., 15 oz. | 0 | 0 | 32 | 0 | 0 |
| 92 | 3 lb., 3 oz. | 0 | 0 | 5 | 0 | 0 |
| 3 | 3 lb., 4 oz. | 0 | 0 | 0 | 0 | 0 |

When kamala and tetrachlorethylene were given simultaneously, only 61.8 per cent of the tapeworms present were removed, while at the same time the combined drugs were only 28.5 per cent effective against the roundworms present. Apparently the combined drugs had no effect on cecal worms (birds 38, 19 and 43).

## EFFECT OF KAMALA ALONE

One gram of kamala was given to each of 15 chickens which varied from 1 pound and 15 ounces to 4½ pounds in weight (table II).

Of these 15 chickens, 7 passed no worms nor showed any, postmortem; 1 was infested with roundworms and tapeworms, 3 with roundworms only and 4 with tapeworms only.

They were destroyed at intervals of 4 to 11 days after dosing. During the time they were under observation none of these chickens showed any untoward symptoms, except in a few instances where a slight purgation was noted during the first 24 hours after dosing, even though six of them (30, 90, 17, 9, 82 and 3) were later found to be tuberculous. No postmortem lesions thought to have been due to the anthelmintic were found in any of the chickens.

As a result of these few tests, kamala in 1-gram doses was found to be 100 per cent effective against tapeworms, and 0 per cent effective against roundworms and cecal worms (bird 30).

TABLE III—*Effect of tetrachlorethylene (1 cc) and kamala (1 gm.) at intervals of 3 days*

| Bird | Weight | Worms Passed | | | | Worms Postmortem | | |
|---|---|---|---|---|---|---|---|---|
| | | After Tetrachlorethylene | | After Kamala | | | | |
| | | Round | Tape | Tape | Round | Round | Tape | Cecal |
| 46 | 2 lb., 12 oz. | 7 | 0 | 25 | 0 | 0 | 0 | 0 |
| 26 | 3 lb., 12 oz. | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 87 | 2 lb., 6 oz. | 10 | 0 | 0 | 0 | 0 | 0 | 34 |
| 18 | 3 lb., 4 oz. | 0 | 0 | 5 | 0 | 0 | 0 | 0 |

## Effect of Tetrachlorethylene and Kamala at Intervals of Three Days

The four chickens (2 pounds, 6 ounces to 3¾ pounds in weight) used in these tests (table III) were given each 1 cc of tetrachlorethylene followed in three days by 1 gram of kamala.

During the three days following treatment with tetrachlorethylene, no toxic symptoms were noted. During the first 24 hours of this period bird 46 passed 7 roundworms, while bird 22 passed 10 roundworms. No tapeworms were passed by any of these chickens during these three days. All of the birds of this group showed the purgative effect of kamala within 24 hours after treatment with this drug.

Birds 18 and 87 died about 24 hours after treatment with kamala, the former having passed 5 tapeworms within twelve hours after treatment. Autopsy of bird 18 showed generalized

tuberculosis, while death of bird 22 was due to roup. No doubt in both instances death was hastened by the action of the anthelmintics.

In the chickens used, 1 cc of tetrachlorethylene, followed in three days by 1 gram of kamala, caused the removal of all of the roundworms and all of the tapeworms. Apparently, neither drug had any effect on the cecal worms found in bird 87.

## Summary

In the few tests made, kamala, in doses of 1 gram to chickens of various sizes, was 100 per cent effective against tapeworms. It apparently had no action on the roundworms which were present in some of the chickens.

Its efficiency against tapeworms seemed to have been reduced when given simultaneously with tetrachlorethylene. A reduction in efficiency of the latter drug against roundworms also was apparent, due possibly to the extremely rapid elimination in chickens (most of the worms having been passed in less than 24 hours), which was no doubt enhanced somewhat by the purgative action of kamala.

However, when the two drugs were given separately at intervals of three days, tetrachlorethylene was 100 per cent effective against roundworms, while kamala removed all of the tapeworms. Neither drug had any apparent effect on cecal worms.

It would seem, therefore, that for best results from anthelmintic treatment of poultry each group of parasites should be treated specifically.

While the loss of a few fowls suffering from tuberculosis, coccidiosis or roup, after treatment of an infested flock, might not be of great economic importance, yet the loss of a few birds suffering from these conditions in these experiments impresses one with the fact that caution should be used when giving anthelmintics to animals suffering from acute infectious or chronic debilitating diseases.

## Conclusions

1. From the results obtained following the administration of kamala in 1-gram doses to the few chickens in these experiments, it can be concluded that this drug is an efficient teniacide and is not toxic for birds weighing from 1 pound to 6½ pounds. The slight purgative action which the drug possesses does not seem detrimental to its use in poultry.

2. When 1 cc of tetrachlorethylene is given simultaneously with 1 gram of kamala to chickens, the efficacy of the latter as a teniacide seems to be reduced considerably, as does the efficacy of tetrachlorethylene for roundworms.

3. Given at intervals of three days, tetrachlorethylene in doses of 1 cc and kamala in doses of 1 gram seems to be effective in removing roundworms and tapeworms from chickens.

4. It would seem that best results would be obtained, in chickens with mixed internal parasitic infestation, by specific medication for each group of parasites.

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[2]Beach, B. A., & Warren, D. M.: Teniacidal value of certain drugs for chickens. Jour. A. V. M. A., lxix (1926), n. s. 22 (4), pp. 498-505.

[3]Schlingman, A. S.: Miscellaneous tests of tetrachlorethylene, a new anthelmintic. Jour. A. V. M. A., lxviii (1926), n. s. 21 (6), pp. 741-754.

## THE EFFECT OF B. SUIPESTIFER IN VIRUS ON RESULT OF SIMULTANEOUS INOCULATION

Hog cholera virus, when first obtained by bleeding infected pigs, at times contains considerable numbers of *Bacillus Suipestifer*. Previous experiments have shown that this organism considerably enhances the disease-producing power of blood from sick pigs. During the year there were vaccinated, in a comparative way, 232 pigs—127 receiving virus containing *B. suipestifer* and 105 with virus from which that bacillus was absent. All the pigs which survived the vaccination treatment were tested later for immunity. There appeared to be no difference in the degree and permanence of the immunity produced by virus containing *B. suipestifer*, on the one hand, and virus from which the bacillus was absent, on the other. The experiments showed, however, that vaccination virus containing large numbers of *B. suipestifer* at the time of injection was liable to produce a so-called vaccination break. In view of these results a study was made of the length of time *B. suipestifer*, when present naturally in the blood of hogs sick with cholera, might remain alive after the addition of 0.5 per cent phenol to the defibrinated blood of such pigs. It was found in the cases studied that *B. suipestifer* in the virus blood was killed within eight days after addition of the phenol, or, more correctly speaking, its capacity to grow or multiply in artificial media was completely inhibited after eight days' exposure to 0.5 per cent phenol.

—*Report of the Chief of the Bureau of Animal Industry.*

# ON THE ROLE PLAYED BY DRAGONFLIES IN THE TRANSFER OF PROSTHOGONIMUS

*By* A. KOTLAN *and* W. L. CHANDLER

*Department of Bacteriology, Michigan State College*
*East Lansing, Michigan*

In 1925 we briefly reported[1] on a newly-recognized fluke disease—prosthogonimiasis—of fowls in the United States. At the time of the discovery that this disease was not confined to migratory water fowls but occurred as well in chickens having access to the lake, the writers visited the locality from where the sick birds came for the purpose of attempting to locate a possible intermediary host of the flukes causing this disease. As mentioned in our first article, the birds were kept on the shore of a small lake and had free access to the water in which different insects, snails, frogs and the like were present in abundance and thus one or the other kind of these animals was suspected to harbor some of the larval stages of our flukes. Therefore, a large number of these insects and snails was collected and thoroughly examined for larval stages of flukes.

In examining different snails, one kind, apparently belonging to the genus Limnaeus, was found heavily infested with larval stages of the fluke genus Echinostomum. Several specimens of these snails contained larval stages of a paramphistomid fluke. No larval flukes showing any relation with Prosthogonimus were found in these snails.

Among the different insects collected at this locality special attention was paid to the larvae (naiads) of dragonflies as well as mayflies, of which a large number was found and collected on the shore of the lake. There were at least four different species of dragonfly naiads present. In examining these insects we discovered in the abdominal cavity a number of encysted larval flukes (metacercariae), the anatomical features of which seemed to reveal certain similarities to those of the adult Prosthogonimus. We decided, therefore, to carry out some experiments in feeding to chickens about twenty to twenty-five dragonfly-naiads representing about four different species.

Received for publication, October 22, 1926.

The results of our feeding experiments were as follows:

Bird 110 received six naiads on May 18. This bird went off egg-production on June 2, although it was fat and heavy and appeared in excellent condition. It was killed on June 25. On postmortem examination the peritoneum was found covered with a thin coating of dark, mottled, brownish material, composed of albumen material filled with fluke eggs. The oviduct contained a much-cracked, thin-shelled hen's egg which was likewise coated with brownish material proving to be composed of fluke eggs. Seventy-six flukes were found on the mucosa of the oviduct and there was evidence that others had decomposed.

Bird 103 received, on May 18, a single naiad differing in shape from those fed to the above-mentioned bird, apparently belonging, however, to the same dragonfly genus. This bird went off egg-production on June 14. No other symptoms were noticed so far as the health of the bird was concerned. The bird was killed on June 26. On postmortem examination the abdominal cavity showed specimens of flukes present in the oviduct, which also contained masses of albuminous material.

Bird 111 received two dead-"pink-eyed"-naiads on May 18. The bird continued laying eggs until June 27, when it was killed. On postmortem examination seven flukes were found in the oviduct.

Bird 104 received five dragonfly-naiads. This bird went off egg-production on May 27. It was kept, however, for further observation and began laying eggs at the beginning of August. The eggs were narrow and elongated, showing numerous constricted areas indicating a pathological condition of the oviduct. The bird never showed any external signs of sickness. It was killed on September 1. No flukes were found in this bird, but some fluke eggs were found on the peritoneum. The wall of the oviduct was thickened.

Birds 101, 106 and 107 received a number of mayfly larvae and different snails. No changes or symptoms were noticed. The birds were killed on July 27. On postmortem examination all were negative.

As shown by the above results, four out of five birds fed with naiads of dragonflies became infested with Prosthogonimi, the infestation being connected in most cases with the development of peculiar pathological changes of the oviduct. It seems unnecessary to call attention to the fact that the flock from which

the birds were selected for this experiment was without any doubt free of a Prosthogonimus infestation.

It is certain, therefore, that dragonflies, especially naiads, play a very important part as intermediary hosts of these flukes. This is substantiated by the fact that the larval flukes (metacercariae) which we found in the dragonfly larva belong to Prosthogonimus. A brief report of these findings was published in the annual report of the Veterinary Division of Michigan State College, for the year ending June 30, 1925.

These experiments were repeated during the spring of 1926. One of us (Chandler) visited Duck Lake on May 27 and again collected various invertebrates, including numerous dragonfly naiads and some newly-emerged adult dragonflies, and cases from which the flies had just emerged. On May 28 one bird "white Leghorn" female was fed a single larva, belonging to the genus Tetragoneuria. This bird was killed June 4, and seven large flukes, undoubtedly belonging to the same species of Prosthogonimus that we are dealing with, were found in the oviduct. No small flukes were found.

On May 28 another female bird was fed six dragonfly larvae. This bird was killed June 21, and thirty-six flukes belonging to the same species of Prosthogonimus were found in the oviduct. These, however, were only about one-half the size of those that were found in the case of the bird harboring seven flukes.

On May 28 another female bird was fed twelve newly-emerged adult dragonflies. This bird was killed June 23 and the oviduct was found to contain ninety-seven flukes, belonging to the same species of Prosthogonimus. These flukes were for the most part only about one-third the size of those in the case of the bird which harbored only seven flukes. Some of them were very small and a few were large.

On May 28 another female bird was fed twelve dragonfly larvae. This bird was killed June 23 and was found to harbor eighty-nine flukes, for the most part only about one-third grown.

On May 28 another bird was fed seven adult dragonflies. This bird, killed June 23, harbored fifty-six flukes.

On May 28 another bird received seven male adult dragonflies, belonging to the genus Tetragoneuria. This bird was killed June 23 and was found to harbor fifty-two flukes.

On May 28 a male bird was fed six dragonfly larvae belonging to the genus Tetragoneuria. This bird was killed June 23. No

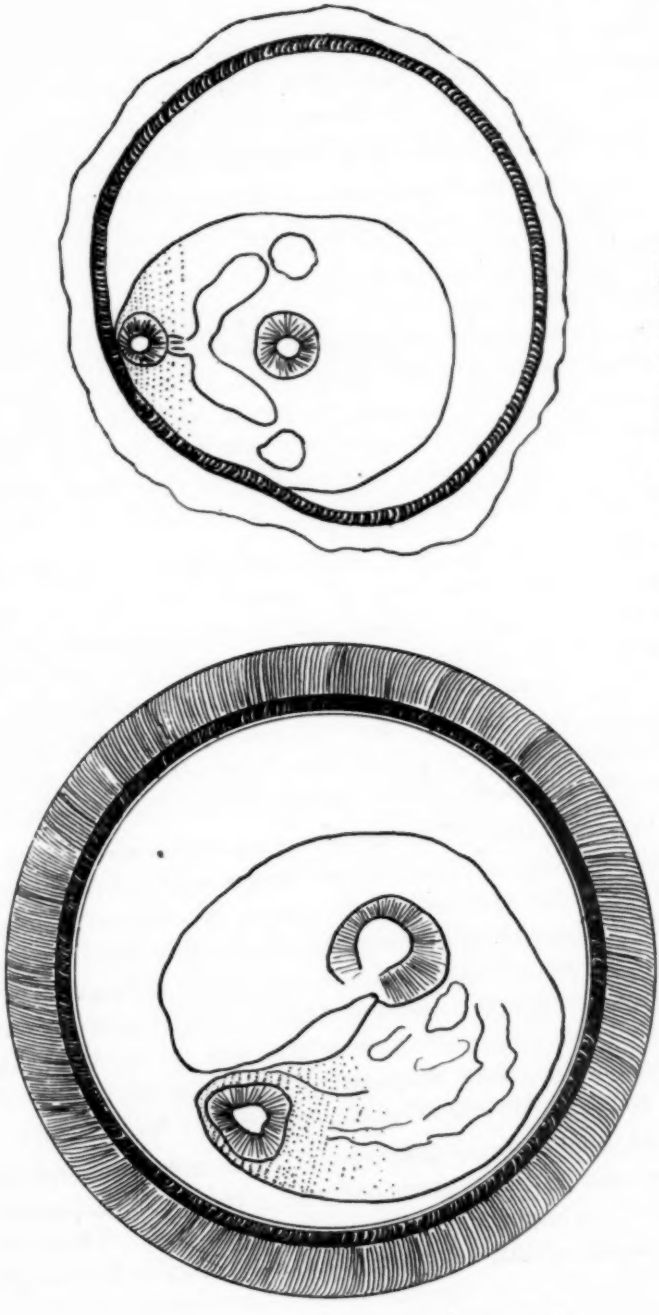

FIG. 1. Drawings of two kinds of metacercariae found in the abdominal cavity of dragonflies.

trace of this bird having been parasitized was found anywhere in the bird.

On May 28 another flock of birds was fed the empty cases of dragonfly larvae. These birds were killed on June 23, but were found not to have been parasitized with the flukes.

On May 28 also, other birds were fed snails belonging to the genus Limnaeus in abundance. These birds, killed June 23, failed to show any indications of having been parasitized by our fluke.

It would appear, therefore, that the dragonfly, both in the larval and adult stages, harbors the infective stage of this fluke. The metacercariae of this fluke were found by microscopic examination in the abdominal cavity of both dragonfly larvae and adult dragonflies. No trace of them was found in the wall of the empty cases. It is of interest to note that when the flukes were present in large numbers their development was retarded.

The chickens on the farm from which these dragonflies were obtained had been kept from the water's edge during the previous year. Only a few Mallard ducks were present in the lake. The extent of the infestation in the dragonflies in that vicinity was greatly reduced as compared with the preceding year. For instance, the previous year one bird receiving a single dragonfly larva harbored thirty-seven flukes, while this year one bird receiving one dragonfly larva harbored only seven flukes.

The cysts containing the larval flukes are of white color and of almost regularly circular outline measuring 0.12 to 0.68 mm. in diameter. The cyst-wall in the larger specimens is about 50 to 67$\mu$ thick, consisting apparently of a double layer, of which the outer one is more transparent and finely striated, whereas the inner one, measuring about one-third of the former, is darker in color and less transparent on account of its dense striation. The smaller cysts show only one layer, agreeing in structure with the narrow, densely-striated layer of the larger cysts.

On account of these differences it seems not impossible that there were two different kinds of cysts present containing metacercariae of perhaps two different fluke species. There is no question, however, but that one of these belongs to Prosthogonimus.

REFERENCE

[1]Kotlan, A., & Chandler, W. L.: A newly recognized fluke disease (prosthogonimiasis) of fowls in the United States. Jour. A. V. M. A., lxvii (1925), n. s. 20 (6), pp. 756-763.

# CLINICAL AND CASE REPORTS

**(Practitioners and others are invited to contribute to this department reports of unusual and interesting cases which may be helpful to others in the profession.)**

## TONGUEWORM INFESTATION OF DOG

*By* D. J. MEADOR, *Selma, Ala.*

Recently a pointer bitch came in for treatment. One day a large female specimen of this parasite (*Linguatula serrata, L. rhinaria, Pentastoma taenoides*) was found in the feed-pan. Autopsy later revealed the presence of four more parasites, which were removed with difficulty from the ethmoid bones.

About five years ago I recovered one of these parasites from a young pointer, but did not report it at the time. The rarity of this species of parasite in this country prompts me to report this.

Hall states that this is not a true worm, but a degenerate of the spiders, ticks, etc. It lives as an adult in the nostrils of the dog and some other animals, and in this stage it is a worm-like animal with external ring-like segmentation. The male is about four-fifths of an inch (18 to 20 mm.) long and the female is about three to four inches long. The eggs from the female worms in the nostrils of the dog pass out in the mucus when the dog sneezes or are swallowed and pass out in the feces. When these eggs are swallowed by suitable host animals eating contaminated vegetation, as by herbivores in grazing, the eggs hatch and the larvae make their way, as a rule, to the liver, lungs, and lymph-glands and there develop to the infective stage.

In the South these larval tongueworms are fairly common in cattle; in Europe, sheep are the most common intermediate hosts. In view of the fact that the larvae may occur also in man, and that the adult is reported from man in one case, this parasite must be looked on as dangerous.

Up to the present time it has been found in the dog only in Georgia, but it undoubtedly has a much wider distribution in this country, as the presence of the larvae in cattle shows. Failure to find it is largely due to failure to look for it.

*Symptoms:* There may be a discharge from the nostrils. The dog sneezes, or there may be loud breathing. The dog will rub

its nose against objects or with his paws. In my opinion there is no connection between this parasite and the so-called "running fits" or "fright disease." Autopsy has failed to find this parasite in several cases of the above disease, although one veterinarian treats for this parasite in treating "running fits."

The owner of the dog under discussion stated to me that he had not fed raw meat at all and had owned the dog for about two years. This dog ran at large and has had access to carcasses of cattle in pastures. He had a discharge from the right nostril but no other symptom.

*Treatment:* Successful treatment will be difficult and will call for trephining and irrigating the nasal cavity.

## SAFETY-RAZOR BLADE RECOVERED FROM STOMACH OF DOG

*By* E. E. Patterson, *Detroit, Mich.*

On November 1, 1926, a client presented an Airedale dog, about ten months old and weighing in the neighborhood of fifty pounds. He appeared to be in good, strong, vigorous condition and the owner related the following history:

"My dog swallowed a safety-razor blade this morning. I tried to get hold of it with my fingers when he had it in his mouth, but before I could do so he had swallowed it. When I made an attempt to open his mouth, he apparently gulped it down. I was frightened almost to death and did not know what to do. Then I thought of your hospital and immediately put him in my car and rushed him over here."

The client was very much excited and my first thought was that we would have to get a physician for her. However, we quieted her and then turned our attention to the dog. The question presented itself: What quantity of food did the dog have in his stomach? Upon being informed that he had eaten a hearty breakfast, some of our anxiety was removed. The next step was to introduce about ten ounces of normal saline solution into the stomach with a stomach-tube. Then 1/10 grain of apomorphin was administered hypodermatically. Then gastro-intestinal lavage was resorted to. All material was removed from the rectum and then a quantity of saline solution was forced through the small intestine and the stomach and out through the mouth. In the process the patient vomited

about a quart of semi-solid material. In the mass we found a Gillette safety-razor blade, as well as a number of roundworms. The dog was returned to the owner in about three hours, none the worse for his experience.

## COD-LIVER OIL AND DAIRY COWS

A writer in *The Country Gentleman*, for December, directs attention to the fact that all over the country there has been a great deal of interest in the question of feeding minerals to cows. The minerals that have been worked with are calcium and phosphorus. Closely associated with the question of calcium has been that of its assimilation, as influenced by cod-liver oil. It is pointed out that the results of investigations show that the feeding of cod-liver oil to dairy cows does not increase the assimilation of calcium. Prof. E. B. Hart, of Wisconsin, and Dr. E. B. Meigs, of the United States Department of Agriculture, at Washington, so report. Neither of these investigators could find that cod-liver oil has any beneficial effect on the mineral nutrition of dairy cows. The article states:

"This would seem to indicate that cod-liver oil alone, and cod-liver oil particularly as mixed in mineral feeds, is without value.

"Prof. H. H. Wing, in New York, has fed cod-liver oil liberally, the past year, to heifers previous to their first calving, in an endeavor to put them in better shape and to eliminate breeding diseases and troubles.

"Apparently here, too, it is completely without effect, except that perhaps their coats were a little smoother. Cod-liver oil is expensive and I think from these experiments that we can conclude that it has no place in the dairy ration."

## ILLINOIS VETERINARIANS STUDY POULTRY PROBLEMS

The University of Illinois held a three-day conference on poultry diseases at Champaign, November 18-19-20, 1926. Special attention was given to demonstrations of the pullorin test for bacillary white diarrhea. Avian tuberculosis also received a great deal of attention, the subject having been presented by Dr. T. S. Rich, B. A. I. inspector-in-charge of tuberculosis eradication in Michigan. Mr. S. J. Stanard, of the State Department of Agriculture, State Veterinarian Laird and Prof. H. R. Smith, of the National Live Stock Exchange, Chicago, assisted the members of the University faculty in putting on an interesting program.

# REVIEW

PRAKTIKUM DER TIERARZTLICHEN GEBURTSHILFE, 3. AUFLAGE. Dr. med. vet. Friedrich Lindhorst, Veterinärrat, Amsttierarzt in Delmenhorst, und Dr. med. vet. Fritz Drahn, a.o. Professor, Prosektor des anatomischen Instituts der Tierärztlichen Hochschule Berlin. viii + 211 pages with 148 illustrations and 1 plate in colors. Richard Schoetz, Berlin, 1924.

This is the third edition of this book and although it follows the second edition within a year and a half it has been essentially re-written in order that due consideration could be given to the more recent literature. At the same time, thirty additional illustrations were added, increasing the number to 149, nearly all of which are original.

Obstetrical practice as it is applied to the parturient female and the fetus is comprehensively yet concisely presented. After describing the dress of the obstetrician and the preparation of his arms and hands, the useful and necessary instruments, the position and preparation of the mother, and the methods of examining the fetus to determine the treatment required, the authors then discuss the abnormalities and pathological conditions of the genital passages and the abnormal positions and the diseases of the fetus which may interfere with parturition, and describe the technic of the various procedures used to effect delivery.

The subject matter is arranged in two parts, one devoted to the cow and mare and the other to sheep, goats and swine. The bitch is not considered. There is also an appendix in which the anatomical essentials and the fundamentals of the mechanics of parturition receive due consideration.

The book is remarkable for the numerous and excellent drawings which illuminate and clarify the text, making it easy for the reader to comprehend the descriptive matter. Malpositions of the fetus and the technic of corrective measures especially are well illustrated. Practitioners should find the work a convenient guide to obstetrical practice and it should also facilitate the study of the subject by the undergraduate. It is printed on paper of better quality than has usually been used in German books in recent years and the letter press is excellent. The book is bound in linen.

# ABSTRACTS

VIRULENCE OF BLOOD IN RABIES. O. A. German. Profilakticheskaya Meditsina, v (1926), pp. 1-176. Abst. in Cur. Med. Liter., Jour. Amer. Med. Asso., Oct. 30, 1926, pp. 15-24.

Dr. O. A. German, of Kharkov, Russia, has demonstrated by biological tests that the blood of man, rabbits and guinea pigs affected with, or dying of, rabies is infectious. He injected a rabbit (subdurally) with blood serum from a woman dead of rabies. The animal developed rabies after eight months and died about five weeks later.

Blood was removed from the rabbit a few days before death and the serum injected into the brain of a guinea pig. The latter died of rabies the ninth day. Another guinea pig was injected (subcutaneously) with the hemolyzed blood from the rabbit. It died from rabies twenty-two days later.

Blood serum from the woman was injected into the muscle of another rabbit. There were some mild manifestations for two days, then the animal recovered completely. Seven months later, the same animal was injected in the brain with fixed virus and developed rabies after nine days; it died three days later.

Other rabbits injected with the same virus developed rabies after three to five days and died on the sixth and seventh day after the injection. Although the experiments showed the virulence of the blood to be low, vaccination is suggested for persons in whom blood from animals with rabies has come in contact with abraded skin or mucous membrane.

G. W. R.

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THE EFFICIENCY OF TETRACHLORETHYLENE IN LIVER ROT OF SHEEP. R. F. Montgomerie. Jour. Comp. Path. & Therap., xxxix (1926), 2, pp. 132-33.

Five ewes affected with liver rot were given tetrachlorethylene in soft gelatin capsules in doses varying from 1 to 5 cc. No toxic symptoms were noted. As a result of these tests it was apparent that tetrachlorethylene does not possess an anthelmintic action towards the common live fluke comparable with that of carbon tetrachlorid. The author is of the opinion that even though the small series of experiments recorded does not justify the conclusion that tetrachlorethylene is of no value in the treatment of liver rot, the results obtained do not encourage further investigation of its value in this connection.

S. S.

# ARMY VETERINARY SERVICE

## CHANGES RELATIVE TO VETERINARY OFFICERS

### Regular Army

Captain Elwood L. Nye reported for duty in the office of the Surgeon General, Washington, D. C., relieving Captain Ralph B. Stewart under orders to proceed to Fort Riley, Ks., for duty.

### Reserve Corps

*New Acceptances*

Second Lieutenants:
Bowers, Joseph Mitchell.....1256 High Street, Pittsburgh, Pa.
Jones, John Daniel,.....Box 541, Nacogdoches, Texas.

*Transfers*

Second Lieut. Joseph H. Ryland, Camden, Alabama, transferred from Infantry Reserve, effective Nov. 15, 1926.

*Promotions*

Helfand, Louis Israel, 325 Pine Street, Philadelphia, Pa., to Captain.
Hodgson, Harold B., 130 Hall St., Athens, Ga., to First Lieut.
Ikard, Wm. Lewis, Box 301, Jerome, Idaho, to First Lieut.

*Separations*

Rowe, Hansford Herndon, Capt. Failed to accept reappointment.
Johnson, Clarence Herbert, 2nd Lieut. Died May 11, 1926.

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## MINNESOTA PROGRAM STRESSES HOG CHOLERA

Hog cholera control will be stressed at the twenty-ninth annual meeting of the Minnesota State Veterinary Medical Association, to be held at the Radisson Hotel, Minneapolis, January 13-14, 1927. Another outstanding feature will be a series of addresses by practitioners, all Minnesotans except Dr. T. A. Sigler, of Greencastle, Indiana, who is president of the American Veterinary Medical Association and an active practitioner as well.

Dr. T. P. White, of the Division of Hog Cholera Control, Bureau of Animal Industry, Washington, D. C., will speak on "The Recent Hog Cholera Epidemic." Dr. J. W. Benner, of the N. Y. State Veterinary College, Cornell University, will give an address on "Hog Cholera Vaccination, Breaks and Their Prevention." Victor E. Anderson, assistant attorney general, St. Paul, is down for a talk on "Legal Phases of Animal Disease Control Work."

Dr. W. C. Prouse, of Minneapolis, is president of the Association, and Dr. C. P. Fitch, chief of the Division of Veterinary Medicine, University of Minnesota, is secretary-treasurer.

# COMMUNICATION

## MINERAL SUPPLEMENTS OFTEN NOT UTILIZED

To the Editor:

I was very much interested in the polemical discussion of the mineral nutrient problem by Dr. Koen in a recent issue of the Journal, in which he takes issue with you, both on ethical and scientific phases of the question. The doctor's oracular style will no doubt convince and mislead some veterinary practitioners, but I think that facts developed by men like Dr. E. B. Forbes, Director of the Institute of Animal Nutrition, Pennsylvania State College and presented in his paper at the recent annual meeting of the American Dairy Association, will have more weight with the majority of veterinarians, and certainly so with the great majority of dairymen and cattle breeders.

In a recent number of the *Holstein-Friesian World* (Nov. 6, 1926, p. 1846), Dr. Forbes states that, "during the feeding of mineral-rich or mineral-poor rations, or during the mineral loss or mineral storage, the utilization of calcium compounds fed as supplements to the ration is often entirely absent, and at best is slight and inefficient." And he further states that, "the present evidence warrants the use of mineral feeds only on an experimental basis, and does not warrant the inclusion of mineral compounds in commercial feeds."

These conclusions are presented by a profound student of animal nutrition and based on years of scientific and practical experimentation.

J. W. Connaway.

*Columbia, Mo., Nov. 18, 1926.*

## OKLAHOMA MEETING DATE CHANGED

Dr. C. H. Fauks, acting president of the Oklahoma State Veterinary Medical Association, has announced a change in the dates of the meeting to be held in Oklahoma City this month. The dates originally selected were January 24-25. These have been changed to January 17-18, one week earlier.

A 21-year test at the Ohio Agricultural Experiment Station shows a loss of about 53 cents on every ton of manure allowed to accumulate in the barnyard.

# MISCELLANEOUS

## A VEST POCKET ESSAY ON "PARADOXICAL"

*By* I. K. ATHERTON, *College Park, Md.*

A certain foot-ball coach and scribe recently used the word "paradoxical" in making the prediction that his team would be defeated by Yale. Despite this handicap, the team trounced the Eli's and another Maryland Plan showed to an advantage. It was evident, though, that in using the word this Byrd was endeavoring to elevate the game of foot-ball to the same intellectual heights to which Gene Tunney has boosted pugilism. It must be remembered, however, that while Gene has many imitators, there are very few who have his punch. "Paradoxical," however, has all the ear-marks of being a perfectly good word and, probably like discretion, should be used more often than it is. Webster says that the meaning of "paradoxical" is "inclined to tenets, or notions contrary to received opinions." Very likely he is right at that. He has always had a good reputation for truth and veractiy and we have no reason to believe that he is stringing us in this. One might have been considered paradoxical but he would not have been crazy had he been inclined to the notion that the St. Louis Cardinals would trounce the Yankees; that Gene Tunney would "raise" Jack Dempsey's face; that the Army mule would kick Yale into submission; and that the Navy goat would butt Michigan's aspirations into a cocked hat. Moreover, some apples are ripe when green.

Paradoxical as it may appear, while this country has been entertaining hog cholera for nearly one hundred years, the swine-owners have more erroneous ideas regarding the introduction and spread of the infection than a Bolshevik has of good government. Some of these opinions are: "It just comes;" "it is an act of Providence;" "bees, mosquitos, flies, birds and floods bring it," etc., etc. In fact, practically everything except the bobbed-hair craze is blamed, while the true culprit—the human agency—is left out of the picture. The three principal, if not the only, factors responsible for starting new outbreaks of the disease are: feeding infected pork in garbage, table scraps, kitchen swill, etc.; the introduction of infected hogs into well herds, and the abuse of the double treatment. Deny, if you can, the proof that all these are the work of hog cholera's human

ally. If the swine owners will dissolve this costly alliance and become members in good standing of the S. P. S. S. M. (Society for the Promotion of Sanitary Swine Management), that sad old song, "Ain't It Awful, Mabel?" will soon be forgotten in the rendition of a quick-step funeral march for H. C. Germ.

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## CAUSE AND EFFECT

### A Tragedy in Ten Acts

I

Walt Hereford has just returned from the State Agricultural College, where he has taken a complete course in veterinary work, specializing in cholera vaccination. Walt has been gone nearly two weeks and reports the course as very interesting.

(New Era *Bugle*, April 1, 1926.)

II

J. Quincy Spriggs, head of the local farm bureau, announces the purchase of 100,000 cc of the finest product the Impotent Serum Company, at Podunk City, produces. It will be here next week and farmers are urged to come in promptly for their quota. A full line of syringes and needles has been procured, and an expert from the Extension Department will be here, next Saturday, to teach all men how to use them.

(New Era *Bugle*, May 1, 1926).

III

Dr. George Smith, local veterinary, reports less vaccination than done for years. He predicts much disease this fall. The doctor says that cheap serum, improper diagnosis and administration is always followed by a trail of death among swine. He states that the owner is given a sense of false security and may lose all his pigs before suspecting the real cause.

(New Era *Bugle*, June 15, 1926.)

IV

Walt Hereford, our expert serum vaccinator, has just finished with Sim Carson's herd of 120 head. Sim is jubilant with Walt's work, and says that by using the Farm Bureau's Impotent serum he saved more than $23.00.

(New Era *Bugle*, July 15, 1926.)

V

Much sickness is reported among the hogs in this community. Sim Carson has lost 45 head so far.

(New Era *Bugle*, Sept. 1, 1926.)

VI

Dr. Smith was called to Sim Carson's yesterday. He reports finding but three hogs left out of 120. These were sick and slaughtered. Cholera symptoms were found on the kidneys.

(New Era *Bugle*, Sept. 15, 1926.)

VII

Nate Tickleberry has lost all his hogs, and Andy Snoop has but four left. The First National Bank has foreclosed on Sim Carson's place. Pigs have been dying like flies the past month.

(New Era *Bugle*, Oct. 1, 1926.)

VIII

Walt Hereford was a passenger on today's train for Chicago, where he intends taking up the plumbing trade.

(New Era *Bugle*, Oct. 15, 1926.)

IX

J. Quincy Spriggs has just resigned as head of our Farm Bureau. Mr. Spriggs states that the serious condition of Mrs. Spriggs' health makes it imperative for them to seek another climate.

(New Era *Bugle*, Oct. 25, 1926.)

X

We regret to announce that Dr. Smith, our local veterinarian, who has been here for ten years, states that he has just accepted an appointment with the government meat inspection department. Dr. Smith leaves the first of next month. He states the reason for quitting practice is that so many agencies interfere with the legitimate practitioner he finds it difficult to keep busy. For this reason, he adds, very few young men are taking up that line of work. This leaves our whole county without a veterinary.

(New Era *Bugle*, Nov. 1, 1926.)

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## VETERINARY CONFERENCE AT PULLMAN

Dr. E. E. Wegner, dean of the College of Veterinary Medicine, State College of Washington, announces that, during the week beginning January 10, his institution will hold a conference for the veterinarians of the four northwestern states and British Columbia. Among the veterinarians of national reputation who will contribute to the program are Dr. Jacob Traum, of the University of California, and Dr. J. C. Flynn, of Kansas City, Mo.

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Corn is attacked by 300 different insects.

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## 1926-1927

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# ASSOCIATION MEETINGS

## GEORGIA STATE VETERINARY ASSOCIATION

The twentieth annual meeting of the Georgia State Veterinary Association was called to order for a two-day session, at the Henry Grady Hotel, Atlanta, September 6, 1926, by President W. E. White, of Tifton, who presided.

Shortly after the meeting was called to order a brief intermission was granted so that the members and their ladies could view the passing Labor Day parade. The address of welcome was delivered by Hon. Rogers Winter, executive secretary of the Stone Mountain Memorial Association. Dr. S. F. Stapleton, of Americus, responded. This was followed by a very timely and forceful address by President White, which was enjoyed by all. The meeting was adjourned Monday at noon so that the members could enjoy a trip to Stone Mountain.

At the Tuesday session several interesting papers were presented and were followed by lively discussions. The attendance was good and two new members were elected, Dr. Foy C. Randall and Dr. S. H. Exley, both graduates of the Veterinary Division of the University of Georgia, 1925.

A resolution was adopted giving the secretary authority to collect money for the purpose of prosecuting violators of the State laws regulating the practice of veterinary medicine in the state of Georgia. The newly-elected president was authorized to appoint a committee, with power to act, to investigate unethical conduct on the part of any member. Under this resolution any member found guilty of unethical conduct may be expelled from the Association.

Officers for the ensuing year were elected as follows: President, Dr. R. M. Walsh, Atlanta; vice-president, Dr. J. L. Ruble, Quitman; secretary-treasurer, Dr. P. F. Bahnsen, Americus.

The next meeting will be held early in November, 1927, jointly with the Southeastern States Veterinary Association and the Florida State Veterinary Medical Association, at Jacksonville, Fla.

PETER F. BAHNSEN, *Secretary.*

## KEYSTONE VETERINARY MEDICAL ASSOCIATION

The regular meeting of the Keystone Veterinary Medical Association was held October 27, 1926, at the University of Pennsylvania School of Veterinary Medicine.

The election of officers for the coming year resulted as follows: President, Dr. William J. Deegan, Camden, N. J.; vice-president, Dr. H. P. Eves, Wilmington, Del.; secretary-treasurer, Dr. C. S. Rockwell, Philadelphia, Pa. Board of Directors: Drs. George A. Dick, E. L. Stubbs, J. W. Vansant, Walter G. White and E. T. Booth.

Dr. C. J. Marshall made a motion, which was seconded and carried, that the meeting be a memorial one in honor of our good friend, Dr. J. W. Adams, who died very suddenly on October 22, 1926, from angina pectoris. Each member present had some pleasant little incident to recall in his association with Dr. Adams. Some spoke of the stories he told us at various times; many spoke of the high esteem in which Dr. Adams was held, personally and throughout the profession, and of the sorrow occasioned by his untimely death. His many interesting addresses and discussions before this organization were always thoroughly enjoyed. He was a scholar, a forceful and witty speaker, a loyal friend, an excellent practitioner and one of the best teachers and surgeons the veterinary profession has ever had. It was also unanimously voted that sincere and heartfelt sympathy be extended to Mrs. Adams and daughters.

There being no further business, the meeting adjourned.

C. S. ROCKWELL, *Secretary.*

## NORTHWESTERN ILLINOIS VETERINARY MEDICAL ASSOCIATION

The annual meeting of the Northwestern Illinois Veterinary Medical Association was held at Freeport, Ill., October 27, 1926. Dr. Charles Rosenstiel, president, occupied the chair. His presidential address covered such important subjects as abortion, avian tuberculosis and bacillary white diarrhea.

Dr. Robert Graham, of the University of Illinois, addressed the meeting and told of the experimental work in animal pathology that is in progress at the University.

Tuberculin testing and the hog cholera situation were other topics which were freely discussed. The sale of, distribution to and use of hog cholera virus by others than competent veterinarians was strongly condemned.

At a business session the following officers were elected for the ensuing year: President, Dr. Roy E. Kluck, Freeport; vice-president, Dr. J. B. Baber, Stockton; secretary-treasurer, Dr. B. L. Lake, Sterling; Board of Censors, Dr. H. C. Barth, Amboy; Dr. C. W. Swingley, Freeport, and Dr. T. E. Lotz, Chadwick.

## NORTH CENTRAL OHIO VETERINARY MEDICAL ASSOCIATION

The tri-annual meeting of the North Central Ohio Veterinary Medical Association was held in the Chamber of Commerce assembly room, Mansfield, November 4, 1926.

Dr. Roy F. Leslie, chief meat and dairy inspector of Cleveland, was the principal speaker. Dr. Leslie explained in detail the system of meat and dairy inspection which had been built up in Cleveland, during the eleven years the work has been under his supervision. Dr. Leslie reported that seventy men were now employed, devoting their entire time to food inspection in his City.

A clinic was held at the hospital of Dr. C. C. Wadsworth following the meeting. At 5:30 a chicken dinner was served at the Elks' grill. During the afternoon, the visiting ladies were entertained by Mrs. Wadsworth. They were shown through the reformatory and then joined the gentlemen at the dinner.

Officers of the Association are: President Dr. W. F. Wise, of Medina; secretary, Dr. C. C. Wadsworth, of Mansfield; treasurer, Dr. H. B. Ropp, of Ashland.

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## SOUTHEASTERN STATES VETERINARY MEDICAL ASSOCIATION

The eleventh annual meeting of the Southeastern States Veterinary Medical Association convened at the College of Veterinary Medicine, Alabama Polytechnic Institute, Auburn, Ala., November 8-9, 1926. Dr. J. H. Morse, of Sumter, S. C., in his annual address, made some very striking recommendations covering the work and field of the veterinary practitioner. The Resolution Committee reported and passed a series of resolutions which defined distinctly the field of operation of the veterinary practitioner, and also the work of veterinarians in various lines. This was interesting and valuable, because there are too many agricultural and other laymen trying to take the field belonging strictly to the veterinarian.

Dr. A. L. Shealey, of the University of Florida, gave an interesting talk on "Leeches of Horses and Mules." He thinks he has made a discovery of the real cause of this disease.

Dr. J. H. Coffman read a paper on "White Diarrhea in Poultry and Its Control." Dr. Coffman thinks the work very promising and states that the laboratory and field work should be done

Veterinarians in attendance at eleventh annual meeting of the Southeastern States Veterinary Medical Association, Auburn, Ala., November 8-9, 1926.

by graduate veterinarians; and that all control work in white diarrhea in poultry should also be handled by veterinarians.

Dr. E. M. Nighbert, in charge of parasitic investigations of animals in south Georgia, gave a splendid demonstration of the method of treating and controlling stomach worms in sheep. He also demonstrated the federal method of controlling the ascaris in hogs.

Dr. E. D. King, Jr., city meat and milk inspector of Valdosta, Ga., gave his celebrated stereopticon lecture on meat and milk inspection as it was developed in Valdosta.

Dr. A. G. G. Richardson, of Athens, Ga., read a paper on "Some Anatomical Differences of Domestic Birds and Mammals." Dr. F. D. Patterson read a very interesting paper on some points in feeding, breeding and handling poultry, in order to prevent disease and secure the greatest amount of commercial value out of them. He also told about the proper housing and care of birds and the methods of avoiding nutritional diseases. A demonstration on postmortem examination of poultry was also given by Dr. Patterson.

Dr. T. A. Sigler, president of the American Veterinary Medical Association, delivered an interesting talk on "Practitioners' Problems." He strongly recommended that all the members of the Southeastern States Veterinary Medical Association and all the state associations should be members of the A. V. M. A. and should attend all meetings in order to get the full benefit of the Association. Doctor Sigler also demonstrated his method of operating on a roarer. The operation was done on a mule and was very interesting.

Dr. Benjamin McInnes, of Charleston, S. C., gave a demonstration of his original method of caponizing chickens.

Dr. N. G. Covington held a postmortem on a mule that had been passed as a roarer. Dr. John W. Adams opened the larynx of the mule at the short course held in February, 1926, and found no laryngeal trouble. All the members present examined this mule and tested him out, and various and sundry diagnoses were made but no one accurately diagnosed the trouble. The postmortem revealed an aneurism of the aorta. The mule showed distinct labored breathing with little exercise and also showed a variation in the heart beat. Of course, most of the abnormal heart action came from the effects of the aneurism. This was a most interesting postmortem. Another postmortem was made by Dr. Covington on a sheep that had no normal opening of the

rectum. The rectum opened into the superior wall of the vagina. This case could have been operated on had it lived until the meeting. Several interesting cases were presented at the clinics besides the above. One, a double tenotomy; another, a ventral hernia in a goat; a Holstein bull that had been successfully treated for inability to do service.

An interesting feature of the meeting was a drill staged for the benefit of the Association by the R. O. T. C. class in riding. Capt. Anderson, mounted on a Thoroughbred, was in command. Some forty or fifty students, mounted, went into the regular drill and demonstrated the movements as taught the riding classes in the R. O. T. C.

A banquet was held on the evening of the first day, at which time a number of interesting speeches were made. In fact, a great many of the members were given one- and two-minute topics which were handled with delight to the hearers. On the whole the meeting was one of the best ever held by the Southeastern Association, everybody being well pleased and greatly benefited by the papers and clinics. The next annual meeting will be held about the second week in November, 1927, at Jacksonville, Fla.

The election of officers resulted as follows: President, Dr. A. G. G. Richardson, Athens, Ga.; secretary-treasurer, Dr. John I. Handley, Atlanta, Ga.

C. A. CARY

## NORTHWESTERN INDIANA VETERINARY ASSOCIATION

The annual meeting of the Northwestern Indiana Veterinary Association was held at Plymouth, November 17, 1926. About thirty veterinarians were in attendance. Dr. F. H. Brown, state veterinarian, delivered an address on the eradication of bovine tuberculosis. Dr. N. S. Mayo, of Chicago, spoke of the progress that has been made in veterinary science during recent years. He predicted even greater advancement during the next few years. Three new members were admitted. Officers for the coming year were elected as follows: President, Dr. Roy Wolfe, South Bend; vice-president, Dr. R. C. Julien, Indianapolis; secretary-treasurer, Dr. Link Grigsby, Walkerton.

## HORSE ASSOCIATION OF AMERICA

Seven years of activity have been completed by the Horse Association of America, which observed the beginning of its

eighth year with an annual meeting and banquet at the Blackstone Hotel, Chicago, on December 1. More than 300 members and friends attended.

During the day, work of the Association was reviewed by W. S. Dunham, Wayne, Ill., president; William M. Brezette, Indianapolis, treasurer; and Wayne Dinsmore, Chicago, secretary. Their reports showed that there is an increasing demand for good horses and sires in the United States, and that importations of Percherons, Belgians and Shires were greater this year than at any time since 1914.

The talks brought out also that, during the past year, 177 pulling contests were held in 13 separate states with 18 dynamometers, as against three contests held in 1923 with one dynamometer; and that the 1926 contests were witnessed by one million people as against about 20,000 in 1923. The speakers emphasized that pulling contests are stimulating the breeding of the very best draft-horse stock.

It was additionally stressed that use of horses on farms has been greatly encouraged this year through the instrumentality of the Horse Association in promoting demonstrations of big-team hitches, by means of which as many as 18 horses can be driven to all combinations of farm implements with a single pair of lines that go to the leaders only.

A report of Regis Lefebure, special representative of the Association, who for a year has been engaged in survey work among transportation users in New York City, showed that New York bakeries are saving $59,000 in delivery costs for every million dollars worth of bread delivered by horse and wagon instead of electric trucks, and $92,000 for every million dollars worth delivered by horses instead of gasoline trucks. His research has shown, also, that to motorize milk delivery equipment in the horse zone in New York City would increase the price of milk from 15 cents a quart to 17 or 18 cents, to cover additional costs.

Officers of the Association were all re-elected for the coming year. In addition to Messrs. Dunham, Brezette, and Dinsmore, they are: R. Lawrence Smith, New York City, first vice-president; Frank H. Sweet, Kansas City, second vice-president; and William E. Murphy, Philadelphia, assistant secretary.

WAYNE DINSMORE, *Secretary.*

## SOUTHEASTERN MICHIGAN VETERINARY MEDICAL ASSOCIATION

One of the best meetings in the history of the Southeastern Michigan Veterinary Medical Association was held in Detroit, December 8, 1926, with thirty-two members in attendance.

The first order of business was a thorough discussion of the proposal of the Michigan Humane Society to take over the management of the city dog pound. Numerous reasons were advanced to show that such a move would not be in the best interests of all concerned. A motion prevailed authorizing President Schlingman to appoint a committee to draw up a resolution and forward it with a letter to the Detroit City Council. This committee was appointed as follows: Drs. G. Floyd Ewalt, E. E. Patterson and H. Preston Hoskins. The committee drafted the following resolution:

RESOLUTION

WHEREAS, There is a movement upon the part of the Michigan Humane Society to secure control of the dog pound of the city of Detroit, and

WHEREAS, This proposal is now being considered by the Common Council of the City of Detroit, and

WHEREAS, The collection of unlicensed dogs is a strictly public function and one that should not be delegated to a private individual or corporation, therefore be it

*Resolved*, By the Southeastern Michigan Veterinary Medical Association, in regular meeting assembled, at Detroit, December 8, 1926, that it is our unanimous opinion that the collection of unlicensed dogs and the management of the city dog pound should be retained under strictly city control, and further be it

*Resolved*, That if, for any reason, the Police Department does not care to retain the custody of the city dog pound, it be turned over to the Department of Health, and be it further

*Resolved*, That in either event there should be the proper veterinary supervision given to the health and well-being of all dogs while kept in the city pound.

The speaker of the evening was Dr. C. D. McGilvray, principal of the Ontario Veterinary College, Guelph, who came to Detroit at the special invitation of the Association. Dr. McGilvray delighted his audience with an extemporaneous address in which he dwelt upon a number of questions confronting the veterinary profession at the present time, including veterinary education, sanitary control work, publicity, poultry practice, association work and the proper place of the veterinarian in public life. Dr. McGilvray had no panacea to offer that would remedy conditions confronting the profession, but at the same time he made a number of suggestions that would be helpful, particularly for the younger practitioners. A rising vote of thanks was tendered Dr. McGilvray upon the conclusion of his address.

Dr. F. D. Egan, of Detroit, gave a talk on "The Physiology of Digestion." Dr. Egan used the blackboard to assist him in bringing out a number of the points which he emphasized. Particular attention was directed to the digestive apparatus of the dog, for the reason that on an average about fifty per cent of the medical cases treated by small animal practitioners are digestive in origin, in many cases brought about through improper feeding, due to ignorance upon the part of the owners. In his remarks Dr. Egan included such points as the actions of the various enzymes, a comparison of the milk of the cow and the bitch, the use of apomorphin in acute indigestion and taka-diastase in gastro-intestinal disorders.

The meeting concluded with a discussion of some of the points brought out by Dr. Egan, as well as a number of others.

H. PRESTON HOSKINS, *Secretary.*

## CENTRAL MICHIGAN VETERINARY MEDICAL ASSOCIATION

The fifteenth annual meeting of the Central Michigan Veterinary Medical Association was held in Jackson, December 9, 1926. Dr. Fred W. Main, of Albion, presided.

Dr. T. S. Rich, of Lansing, B. A. I. inspector-in-charge of tuberculosis eradication in Michigan, described the progress made during the past year. Dr. Rich reported that only twelve counties have failed to take up systematic tuberculin testing.

Dr. B. J. Killham, state veterinarian, gave a résumé of animal disease control work in the State. Mr. W. J. Antcliff, of Norvell, chairman of the County Board of Supervisors, urged further cooperation between state and local veterinarians in tuberculosis eradication.

Senator-Elect Jay Binning delivered an address on "State Legislation and the Veterinary Medical Profession." The veterinarians were promised support for any worthy legislation sponsored by the profession. Mr. R. E. Decker, agricultural agent of Jackson County, expressed his ideas on the relation of count-agent work to veterinary practice.

Dr. J. W. Patton, of Michigan State College, discussed bacillary white diarrhea of chickens and Dr. A. B. Curtis, of Hillsdale, described the new glucose treatment for parturient apoplexy in cattle.

A dinner followed the meeting, with Dr. B. J. Killham acting as toastmaster. He announced that the next Lake States Tuber-

culosis Eradication Conference would be held in Michigan. Secretary Armstrong immediately extended an invitation for the Conference to be held in Jackson.

An election of officers resulted in the re-election of Dr. Fred W. Main as president, and Dr. W. N. Armstrong, of Concord, as secretary-treasurer. The next meeting of the Association will be held in Jackson, in May.

## BEXAR COUNTY VETERINARY MEDICAL ASSOCIATION

The Bexar County Veterinary Medical Association met at the Chamber of Commerce, San Antonio, Texas, December 16, 1926. The principal speaker of the evening was Col. M. L. Crimmins, U. S. A. retired, who addressed the Association on "Poisonous Snakes and the Treatment of Their Bites." The meeting was attended by a number of members of the medical profession and their wives.

U. E. MARNEY, *Secretary.*

## STUDIES ON SOURCES OF TUBERCULOSIS INFECTION

People are more likely to contract tuberculosis from inhaling infected dust than they are through encounters with coughing patients in the active stages of the disease, according to Dr. Friedrich Neufeld, director of the Koch Institute at Berlin. Physicians present at the annual meeting of the National Tuberculosis Association held recently heard Dr. Neufeld describe his experiments to ascertain the most significant sources of tuberculosis infection. More "Spitting Prohibited" signs would seem to be the moral of the German scientist's results. His researches indicate that dust, either breathed in or picked up on the hands and containing dried-up but active germs, causes more tuberculosis than inhaling the droplets from the cough of a tuberculosis patient. Many persons may have the disease in a latent form and be quite unaware of the health menace they present to the world at large.—*Science.*

Some rush to the ends of the earth for happiness; others find it in back-yard gardening.

Don't wait for great opportunities. Seize common, everyday ones and make them great.

# NECROLOGY

## WILLIAM STUBBS

Dr. William Stubbs, of Caledon, Ontario, died November 28, 1926, following an illness of three weeks. He was 82 years of age.

Following his graduation from the Ontario Veterinary College, in 1869, Dr. Stubbs located in Caledon and established a large practice. He soon became deeply interested in politics. In 1895 he was elected a member of the Dominion Parliament, the first veterinarian to be elected to this body. He retired from Parliament in 1901 and was appointed Inspector of Animals for Ontario, a position he held until a few years ago.

Dr. Stubbs' hobby was hunting. He organized the Caledon Hunt Club and was its president for many years. He is survived by two daughters and one son.

Up until the time of his death Dr. Stubbs was the oldest living graduate of the Ontario Veterinary College, a distinction which now passes to Dr. Joseph Hawkins, of Detroit, Mich., a member of the class of 1871.

## ARTHUR DWIGHT HUBBELL

Dr. A. D. Hubbell, of San Bernardino, California, died December 1, 1926, aged 57 years. He had been in poor health for some time, but was feeling better shortly before his passing, so much so that at the time of his death he was on a quail hunt. When complaining of fatigue he sat down to rest, passing away very suddenly.

A native of Connecticut, Dr. Hubbell was graduated from the Chicago Veterinary College in 1906. The same year he went to California and located in Los Angeles, where he conducted a large practice for a number of years. He later removed to San Bernardino, where he had acquired some property interests. He was County Veterinarian until 1925, when he resigned.

Dr. Hubbell joined the A. V. M. A. in 1910. He was also a member of the California State Veterinary Medical Association, the Southern California Veterinary Medical Association and the Orange Belt Association. He was an Odd Fellow and a Mason. He is survived by his widow.

## ROY O. WILSON

Dr. Roy O. Wilson, of Dunseith, No. Dak., died July 5, 1926. Born in Wisconsin, August 26, 1888, Dr. Wilson received his veterinary education at the Kansas City Veterinary College. He was graduated in 1915 and joined the A. V. M. A. in 1916. At different times he practiced at Stanley, No. Dak., Choteau, Mont., and Princeton, Minn. Dr. Wilson is survived by his widow and three children.

## CLYDE L. BRAGG

Dr. Clyde L. Bragg, of Loveland, Ohio, died October 15, 1926, in his 45th year. He was a graduate of the Cincinnati Veterinary College, class of 1916, and was a member of the Ohio State Veterinary Medical Association.

## R. S. JACKSON

Dr. R. S. Jackson, a registered non-graduate, formerly of Lawrenceburg, Ind., committed suicide in Indianapolis, November 10, 1926.

## THOMAS HOFER

Dr. Thomas Hofer, a retired veterinarian of Hebron, Ky., died November 29, 1926.

Dr. J. F. Rheinhardt was killed when struck by a train at a crossing in Apple Creek, near Wooster, Ohio, November 3, 1926.

## ENGAGEMENT

Lieutenant-Colonel J. J. Aitken, C. M. G., D. S. O., O. B. E., R. A. V. C., attached to the Veterinary Corps, U. S. Army, 1917-18, and Constance Marion Drake, daughter of Brigadier-General Drake, C. B., of Grazley Lodge, Farnborough, Hants, England, and of the late Mrs. Drake.

## MARRIAGE

Dr. C. B. Erickson (O. S. U. '25), of Audubon, Minn., to Miss Ruth Anna Burriss, September 15, 1926.

## BIRTH

To Dr. and Mrs. M. J. Kennedy, of Missouri Valley, Iowa, a daughter, Patricia Eileen, November 25, 1926.

## PERSONALS

Dr. Bruce Kester (O. S. U. '25) is with the Board of Health, Dayton, Ohio.

Dr. F. A. Hall (Corn. '23) has established himself in practice at Garrett, Ind.

Dr. Horace B. F. Jervis (Ont. '04), of Wassaic, N. Y., has gone to Sawtelle, Calif.

Dr. J. T. Gruber (O. S. U. '08) is city milk and meat inspector of Marion, Ohio.

Dr. C. W. Eddy (O. S. U. '00), formerly of Cleveland, Ohio, is at Guanajay, Cuba.

Dr. K. J. Moye (K. C. V. C. '14) has removed from Hays, Kans., to Keytesville, Mo.

Dr. Charles W. Fisher (Ind. '17) has resumed practice and located in Martinsville, Ind.

Dr. Earl S. Hinkle (Ind. '09), formerly of Markle, Ind. has removed to Centerville, Ind.

Dr. L. F. Vaughn (K. C. V. C. '16) has been transferred from Fresno to Los Banos, Calif.

Dr. L. C. Butterfield (Chi. '02), of Reno, Nevada, was a visitor in Ottawa, Ill., in December.

Dr. Alfred W. Anderson (O. S. U. '26) is practicing with Dr. R. R. Powell, of Cleveland, Ohio.

Dr. L. A. Swenson (Chi. '17), of Sherburn, Minn., reports practice good, with plenty of snow.

Dr. V. A. Holby (U. P. '16), formerly of Somerville, N. J., is now living in New Rochelle, N. Y.

Dr. C. W. Dwyer (Mich. '21), of East Berkshire, Vt., gives Enosburg Falls, Vt., as a new address.

Dr. N. C. Elberson (T. H. '12), of Anderson, Ind., recently completed a new veterinary hospital.

Dr. Fred E. Graves (Chi. '16), of Galesburg, Ill., has opened a modern small animal hospital at 759 East Main St.

Dr. Hulbert Young (U. P. '00), of Baltimore, Md., is manager of the Walker-Gordon Laboratory located in that city.

Dr. C. C. Winegardner (West. '04), of Goshen, Ind., has opened his Riverside Sanitary Hospital on West Lincoln Avenue.

Dr. C. W. McLaughlin (Chi. '17), of Melvin, Ill., has accepted a position with the Western Weighing and Inspection Bureau.

Dr. Serafin Santamaria (Havana '14) has been appointed director of the Biological Laboratories at Marianao, Havana, Cuba.

Dr. A. H. DeGroot (Gr. Rap. '17), of Dundee, Mich., has been appointed Monroe County (Mich.) veterinarian by the Board of Supervisors.

Dr. D. A. Curtis (Gr. Rap. '12), of Breckenridge, Mich., has been appointed Gratiot County (Mich.) veterinarian by the Board of Supervisors.

Dr. C. H. Stevens (U. P. '11), of Stevensville, Montana, was a recent visitor in Philadelphia, renewing acquaintance with classmates and other friends.

Dr. J. E. Laidlaw (Ont. '94), of Bluffton, Ind., received extensive bruises and lacerations when his autompbile crashed into a fence east of the city recently.

Dr. Dayton Warren (O. S. U. '24) has been appointed an instructor in physiology in the College of Veterinary Medicine, State College of Washington, at Pullman.

Dr. F. W. Crawford (K. S. A. C. '23), of Quincy, Ill., has accepted an appointment with the U. S. Bureau of Animal Industry and reported for duty at South St. Paul.

Dr. R. C. Julien (McK. '07), of Indianapolis, Ind., was the chief speaker at the annual meeting of the Carroll County (Ind.) Farm Bureau held at Delphi, December 14.

Dr. Edgar C. Howell (U. P. '12), of Bishop, Calif., has moved to Reno, Nevada, where he is erecting a residence and hospital, with the intention of entering general practice.

Dr. Roberto Plata Guerrero (U. P. '20) has accepted an appointment as veterinarian to the Government of Ecuador. He sailed from New York, the second week in December.

Dr. Moore B. Herron (U. P. '12), of Canonsburg, Pa., recently enrolled for the special course in the diseases of small animals at the University of Pennsylvania School of Veterinary Medicine.

Dr. Angel Iduate (Havana '12), of Havana, Cuba, has been commissioned by the Secretary of Agriculture of Cuba to go to Mexico to study the foot-and-mouth disease situation in that country.

Dr. T. P. Polk (U. S. C. V. S.'11), of the University of Kentucky, addressed a large gathering of poultry and hog raisers at Princeton, Ky., November 23. He discussed poultry and swine diseases.

Dr. W. L. Boggy (Chi.'15), formerly Monroe County (Ill.) veterinarian, has been employed as Randolph County veterinarian at a salary of $3600 a year. Dr. Boggy was scheduled to start his new duties January 1.

Dr. E. E. Wegner (Wash. '08), Dean of the College of Veterinary Medicine, State College of Washington, was elected Mayor of Pulman, Wash., at the recent municipal election. Dr. Wegner had previously served as a member of the city council for a number of years.

Dr. C. J. Mulvey (McGill '94), of Mooers, N. Y., was granted a leave of absence for two months, beginning November 1, by the Franklin County (N. Y.) Tuberculosis Committee. Dr. Mulvey planned to have an operation performed upon his throat and a recent report would indicate that he expected to be able to return to duty January 1.

Dr. T. E. LeClaire (Laval '90), of Calgary, Alta., will leave soon for the South, where he expects spending the winter, returning to Calgary about next June or July by way of New York and Montreal. From Calgary Dr. LeClaire will proceed to Tucson, Ariz., by way of Los Angeles, then to New Orleans, from which port he will sail to Cuba and Jamaica.